DOCUMENT RESUME

ED 319 731 SP 032 377

AUTHOR	Enwonwu, Cyril O., Ed.
TITLE	Impact of Nutrition on Health and Disease in Blacks and Other Minorities. Proceedings of the Meharry Medical College Annual Nutrition Workshop (1st, Nashville, Tennessee, October 28-30, 1987). Annual Nutrition Workshop Series, Volume 1.
INSTITUTION	Meharry Medical Coll., Nashville, Tenn.
PUB DATE	Oct 87
NOTE	235p.
PUB TYPE	Collected Works - Conference Proceedings (021) -- Reports - Descriptive (141)
EDRS PRICE	MF01/PC10 Plus Postage.
DESCRIPTORS	Acquired Immune Deficiency Syndrome; *Blacks; Cardiovascular System; Eating Habits; Genetics; Hypertension; *Medical Research; *Minority Groups; *Nutrition; Obesity; Sickle Cell Anemia; Socioeconomic Status; *Special Health Problems

ABSTRACT

Participants in this workshop were scientists from various disciplines, including public health, oncology, nutrition, epidemiology, biochemistry, immunology, pharmacology, pediatrics, geriatric medicine, and the behavioral sciences. The workshop featured deliberations by medical experts on the dimensions and demographics of hunger in America. The deliberations included papers on nutrition throughout the human life-cycle, interface between dietary habits and some prevalent chronic health problems, nutrition and immunity with emphasis on protein-energy malnutrition, cancer and nutritional management of AIDS, the nutritional implications of sickle cell anemia, and local as well as international nutrition issues. This publication includes 18 papers presented at the workshop as well as discussions stimulated by the addresses. (JD)

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IMPACT OF NUTRITION ON HEALTH AND DISEASE IN BLACKS AND OTHER MINORITIES

EDITED BY
CYRIL O. ENWONWU

Annual Nutrition Workshop Series
Volume 1

CENTER FOR NUTRITION
Meharry Medical College

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Impact of Nutrition on Health and Disease in Blacks and Other Minorities

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Prepared for publication by
The Office of Public Relations
Meharry Medical College

First Annual Nutrition Workshop
October 28-30, 1987

PREFACE

The United States of America enjoys an affluence that unfortunately keeps many of its citizens overfed. Ironically, the country also contains depressing pockets of unacceptable undernutrition. It is now widely accepted that nutrition-related diseases constitute the most pervasive health problems in the US, disproportionately affecting blacks and other minority groups. The dimensions of the problems are still not fully defined. Available data do however indicate that the incidence of low birth weight in blacks is about 1.5 times that for white Americans, and infant mortality rate is almost twice as high in the former than in the latter group. Additionally, the prevalence and severity of such serious health problems as hypertension, stroke, diabetes, obesity and cancer, which have strong nutritional components in their etiology and prevention, are much higher among blacks than in whites. Thorough understanding of the reasons for disparities in the nutrition-related diseases among various racial groups residing in the same country, is an important prerequisite for rational solution of the problems.

Early in 1986, Meharry Medical College, a historically black institution which for more than a century, has been committed to exploring and solving health problems of special concern to the disadvantaged, established a Center for Nutrition. This was made possible by the generous support of the General Foods Fund, Inc. The Center has a conceptual focus addressing mainly but not exclusively, the nutritional problems of blacks and other underserved minorities. A primary activity of the Center for Nutrition is an annual nutrition workshop during which experts review current knowledge of a particular aspect of nutrition related to blacks and other minorities.

The theme of the first Annual Nutrition Workshop, held at Meharry Medical College on October 28 - 30, 1987, was "IMPACT OF NUTRITION ON HEALTH AND DISEASE IN BLACKS AND MINORITIES". Under this broad theme, experts deliberated on the dimensions and demographics of hunger in America, nutrition throughout the human life-cycle, interphase between dietary habits and some prevalent chronic health problems, nutrition and immunity with emphasis on protein-energy malnutrition, cancer and nutritional management of AIDS, the nutritional implications of sickle cell anemia, and local as well as international nutrition issues. A large number of scientists from various disciplines, including public health, oncology, nutrition, epidemiology, biochemistry, immunology pharmacology, pediatrics, geriatric medicine, and the behavioral sciences attended the workshop.

The first Annual Nutrition Workshop was financially supported by the General Foods Fund, Inc., Kraft Foundation, US National Institutes of Health, United States Department of Agriculture, The Philip Morris Companies, The Proctor and Gamble Company and Ross Laboratories. Meharry Medical College is very grateful for their very generous support.

Cyril O. Enwonwu

WORKSHOP TIMETABLE

Wednesday, October 28, 1987

7:30 am Registration, S.S. Kresge Learning Resource Center

Opening Session

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OVERVIEW OF NUTRITIONAL HEALTH OF MINORITIES

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9:40 Nutritional and Lifestyle Disorders in Blacks - James P. Carter 9

10:20 Coffee

10:40 Nutritional Health of Native Americans - M. Yvonne Jackson 17

11:20 Discussion 25

12:00 Lunch

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NUTRITION THROUGHOUT THE LIFE-CYCLE

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J. Clinton Smith

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3:30 Coffee

3:45 Discussion 231

4:15 Closing - Charles W. Johnson, Vice-President for Academic Affairs 235
Meharry Medical College

ADDRESS OF WELCOME

David Satcher, M.D., Ph.D.
President, Meharry Medical College

Dr. Johnson, and members of the Planning Committee for this very important conference on the Impact of Nutrition on Health and Disease in Blacks and other Minorities, I am delighted to be able to participate in this program and to welcome you on behalf of Meharry Medical College. What I would like to try and do in welcoming you is to place this conference in perspective as it relates to our institution and to what we view as a tremendous challenge in this country. First I should say that every year on the first Monday in October we have the opening convocation at Meharry Medical College and a few weeks ago we celebrated our one hundred and twelfth convocation. Throughout its history, Meharry Medical College has targeted the needs of the underserved in this country and it has focused on providing access to professional careers to blacks, other minorities and in many cases, to students who have a commitment to the underserved. Concern for the underserved has been Meharry's mission.

In 1980 the Robert Wood Johnson Foundation did a special study on Meharry and released a report in which among other things it pointed out that at that time, more than forty percent of all the black physicians and dentists practicing in this country were graduates of Meharry Medical College. In addition it found when it looked at graduates over the last ten years before that time, that 75 percent of these graduates were practicing in underserved rural and inner city communities. We feel that with that kind of pattern as it relates to this institution, Meharry has indeed been faithful to its mission. That is only part of the story because you really have to get out there or visit those rural towns in the south where the only physician or dentist is a Meharry graduate. You also have to go into some of the major inner cities of this country like Harlem and Watts and see the very critical role that Meharry graduates have played or are playing to really appreciate the mission of this institution. Additionally you have to go to places like Zambia in Africa where in the last several years they have developed village clinics run without physicians to provide access to health care, especially for women and children. The University of Zambia at the time of my visit in 1986 had twelve village clinics and eight out of twelve clinics were headed by nurses who graduated from Meharry in the 70's. These few examples underscore the significance of our mission as well as the awesome responsibility of all of us here in Meharry today. In the early 1970's under the leadership of Doctor C. W. Johnson, who is the Vice President for Academic Affairs, the institution started a program in graduate studies and research and since that time has graduated approximately one tenth of all blacks in this country who have received Ph.D. degrees in the health sciences. So in addition to the contribution that the institution has made in terms of health services we have also taken very seriously our responsibility to contribute to production of health scientists who would in fact be on the forefront of new knowledge in various areas. I should point out as some of you well know that less than two percent of the faculties in medicine and dentistry in this country are black. There is a very serious problem in terms of the under representation of blacks and other minorities in the sciences in general and it is a disturbing problem that is getting worse.

In 1982 we embarked upon the plan for academic renewal at Meharry and there were four components to that plan. One component is to significantly strengthen our faculty in size and depth. Secondly, we decided to develop research centers in critical areas like nutrition, geriatrics, tropical diseases and others. Thirdly, we wanted to provide more educational support in terms of library services and scholarships for students. Fourthly, there was a need to revitalize facilities at this institution. It was a very successful campaign in that the financial goal was to raise $33,000,000 to support these areas and we raised approximately $38,000,000. The one area that gave us much pleasure was the development of research centers. One of the major grants was from General Foods to develop a Center of Excellence in Nutrition. A major focus of the grant is to provide the kind of program that assures that every student at Meharry whether in the school of medicine, dentistry, graduate studies or allied health, would leave here with a real understanding of nutrition and the impact of nutrition on health and disease in general, and with a special sensitivity to that issue as it relates to blacks and other minorities. Why is it so important to be especially concerned about the impact of nutrition on blacks and other minorities? In October of 1985, then Secretary Heckler released a study from the Task Force on the Health Status of Blacks and Other Minorities that showed among other things that even in 1985 there existed in this country a significant gap in the health status of blacks and other minorities when compared to whites. Infant mortality in 1985 was twice as great in blacks as in whites. Whereas there has been significant progress in this country since 1950 in infant mortality, the gap between blacks and whites continued and the relationship between that and nutrition is something that I'm sure you will be talking about at this meeting. The whole issue of premature birth, low birth weights and a lot of other problems surrounding pregnancy and neonatology and how nutrition impacts upon those problems are timely. The Secretary's report also pointed out the disparity in deaths from cardiovascular diseases with special emphasis on hypertension and I'm sure there will be some discussion of their relation with nutrition. A very disturbing disparity exists when we look at cancer mortality such that the rate of increase mortality from cancer in blacks in recent years has been so much greater than in the rest of the population. Anybody who is concerned about nutrition would have to be concerned about the nutritional implications of cancer morbidity and mortality in this country. The bottom line of Secretary Heckler's report was that every year in this country, sixty thousand more blacks die than would die if we had the same age and sex adjusted death rates as whites. Sixty thousand excess deaths among blacks take place every year in this country. I just hope that as this conference progresses that we will get a better understanding of some of the nutritional problems related to those deaths and how we can best educate students who are the future health care providers in this country and abroad. Now I want to make it very clear that the health status of whites in this country is by no means perfect. So when we talk about the gaps it is not to imply that the health status of whites in this country is perfect.

It is interesting that as I talk about Secretary Heckler's report, the present Secretary of Health and Human Services, Doctor Otis Bowen would be landing at the new airport in Nashville. At the airport he will establish the first AIDS education package in an airport setting and will go from there to St. Thomas Hospital and at noon will be visiting the Meharry campus. I am very pleased that Dr. Enwonwu and the people planning this nutrition conference have, at very short notice, been very cooperative in helping us to welcome Dr. Bowen and to listen to what he has to say especially about the problem of AIDS. At noon time we will be interacting with Dr. Otis Bowen.

As most of you know, the Senate, following the House, has recently passed the catastrophic health package. Even though there is still cause for concern in that area, it is a major first step in dealing with a very serious problem in this country. How do we deal with catastrophic illnesses? The fact is that as a nation we should not be willing to tolerate the situation whereby the elderly in this country lose their entire life work because we don't provide adequate system of health care for them. I view this as an important first step. It certainly is not enough but we have to take first steps and then we have to move on to other steps. The catastrophic health bill as you know is a product of the efforts of Doctor Bowen and hopefully we can encourage him to proceed along that line so that access to health care will be available to all Americans. The other thing of course is that many times, families are destroyed financially because of accidents or birth defects that occur in members of the family. Hopefully in time this will no longer be true and this nation will in fact be prepared to take care of people who suffer those kinds of unfortunate experiences as well as prepare to take care of our elderly through all of the long term care which they will need. So it is an important first step as we welcome Secretary Bowen here today.

Also, we must continue to push to deal with the problem of hunger in this country and throughout the world. I am delighted to welcome Larry Brown from Harvard. I had the opportunity to work with Larry a few years ago during the time that the physician task force on hunger program was taking place. I believe we went to several places together but I remember East Tennessee, looking at hunger in those areas. It is a real pleasure to have Larry here, knowing of his deep commitment to helping to solve the problem of hunger in America.

In summary then, you are at an institution where the nutrition center is very important to our mission and very important to what we are trying to do here. Your presence here today helps to foster that mission and helps to develop the center. We feel we are very fortunate to have been able to recruit somebody of the stature of Cyril Enwonwu to head our Center for Nutrition and in the very short time that he has been here the Center has made significant progress. There is a National Advisory Committee to the Center. I am sure you are going to hear more about that. I think Dr. Carter who chairs the committee is with us and will be speaking during the workshop. There is an Executive Council within the institution which sets guidelines and priorities for the Center. Additionally, there is a program development committee which is involved in the activities of the Center at the operational level. So the organizational structure for the Nutrition Center is a model for Centers of Excellence at Meharry. We welcome all of you. I am sorry I will not be around for all of the sessions but hope to get in as much as possible.

Thank you very much.

FIRST SCIENTIFIC SESSION

Overview of Nutritional Health of Minorities

CHAIRPERSON:

William J. Darby, Jr., M.D. Ph.D
Professor of Biochemistry
and Nutrition (Emeritus)
Vanderbilt University
Nashville, Tennessee

Dimensions of Human Hunger in America

J. Larry Brown
Harvard School of Public Health
Department of Health Policy and Management
Boston, MA

Beneath the picture of growing national prosperity, and behind the facelessness of government statistics, are the dimmed images of American workers on the treadmill of low-wage jobs. The condition of these citizens is living testament to something fundamentally troubling in our nation.

After several years of sustained economic growth, more Americans are living in poverty than when the decade began. Many of them are workers whose earnings frequently come to less than the amount the government says is required to meet minimal needs - $9,000 for a family of three, $11,000 for four. So serious is the problem of low wages that hunger is now reaching into new groups of our population.

When it opened two years ago, the Star of Hope Mission in Houston served only men. That changed quickly as women and children were driven to its refuge by the depressed oil industry. By 1987, some 3.1 million Texans were living in poverty.

Little Tina finished her oatmeal while her father described how she had lost several pounds while the family lived on rice and beans. "We live in hell," the man reported. "Every day I go to the Labor Pool. When I'm lucky I get a day job for $3.80 an hour." This family represents the destitution of the area. More than 320,000 jobs were lost in the Texas oil and gas industry between 1982 and 1986. Some 280,000 homes were on the market at the time of our visit to Houston, testimony to the broken dreams of under-employed families. Local churches and neighborhood agencies see the victimized families on the streets, a new class of social service clients in the making. "They're mostly middle-age and white, "an agency director told us. "They once earned $16 an hour. Now they make about $4.00."

Dramatic increases in emergency food requests overwhelm public and private efforts to feed the hungry. Of the 3.1 million Texans in poverty, just over one million receive food stamps. The rest are left to search for hand-outs. Suzanne Hinds of the United Way reported that "At no time has the need been so great." Senator Hugh Parmer who heads a bipartisan legislative commission on hunger announced that since 1983, emergency food requests have more than doubled statewide. Food banks increased their distribution from 8 million pounds to 50 million a year, but 800,000 Texans go hungry some time each month.

In New Orleans the next day, Dr. Sandra Robinson told of Louisiana citizens suffering from high unemployment, high infant mortality, and low AFDC levels for the destitute. This physician who runs the State Department of Health and Human Resources observed that "there is a new class of poor, and federal policy limits are killing them. People are required to get even poorer in order to get help," she said. "We put them in a bottomless pit." Forty year old Robert Carter came to our public meeting to describe life in this pit. A husband and a father, his family now has no running water and no electricity. While looking for work, he applied for AFDC for their children, but was deemed ineligible because he owned a small piece of property.

"Would you rather help a piece of me or all of me," he asked in reference to federal rules that make him dispose of his assets before qualifying for temporary help.

In another part of the country we found the kind of quiet desperation associated with other nations. Entire families moving from town to town looking for food or a job. Grown men foraging through junk yards, children coming to feeding centers on their own in hopes of a meal.

This is Blackhawk County, Iowa, right in the heart of America's breadbasket. "It's awful," a local grocer told us. "This morning a farmer stood in the checkout line with a loaf of white bread, powdered milk and two cans of dogfood. I looked him in the eye and he turned red and walked away."

Much has been written about the death of the family farm, but when a farm dies so does much of the community. The industries that support farming fail too, such as the local John Deere tractor plant that laid off 8,000 workers. It is interwoven misery somehow representing the destruction of a way of life.

A nurse at the health clinic in Waterloo said that she and her colleagues wondered where all the people are coming from who ask for food at her medical facility. "We soon realized that most of them are middle age farm couples with children." It is dramatic to face hardy men and women who until recently enjoyed relative affluence but now have virtually nothing. It is startling to drive into Waterloo, surrounded by fields of corn, to find a van with the words "food bank" painted on the side. But feeding the hungry has become serious business in the Midwest.

"Last summer we offered a feeding program for our children," a local school official told us. "We expected 300 children to attend. But more than 2,000 came the first day. We thought we knew our community, but we were in for a big surprise." It is an embattled and often bitter population, these independent-minded Midwesterners. They say things like "you can't believe government any more," or something to the effect that "a man's word is no longer any good." Hungry and needy for the first time ever, they face the overwhelming obstacles more familiar to other destitute citizens.

"The government makes you spend yourself into dirt," pointed out a 53 year-old farmer who looked at the floor to conceal the moisture in his eyes. "You gotta get rid of everything just to qualify for $60 in food stamps to feed your kids. It ain't right." The man's family recently lost their farm, the only home they had ever known. The teenage daughter watched her father walk alone through the fields as her mother tried to console her: "Honey, he's had this land longer than he's had you. It's like losing a child, he needs some time to be alone."

In Minnesota one finds an image of relative affluence, a decent standard of living for hard-working families. Yet, nearly half a million of its citizens live in poverty, an increase of 100,000 since the end of last decade. About half this increase has been among two-parent families. Sandra Gardebring, State Human Services Commissioner, reported that hunger is serious in areas of Minnesota. At the emergency fund in Marshall, volunteers are unable to keep up with the growing need. The program gave out 360,000 pounds of food last year. That number will double this year.

"Our food crisis," noted one worker, "is now a chronic nightmare." Marshall is one of those small towns the rest of America sees on television. Located in the southwestern part of the state where no town is over 20,000, it is a picture of tranquility. But hidden from the outsider is the reality of tragedy. "It's hard to see who's hungry here," admitted the mayor, "but for many of our families it's the last year on their farm."

Local officials report that federal regulations prevent their feeding many in need. "Food stamp rules totally hamstring us, "reported welfare director Frank Morris. "People in their fifties are hungry and aren't eligible for anything."

In nearby Lincoln County we traveled a

checkerboard of rural roads to the frame house of a farm family where middle-age couples had agreed to meet with us. Not the kind of people to express emotion easily, two of the men lost their composure as they talked. "I been here all my life," observed one ruddy farmer who wiped his eyes with huge fingers. "But if you come back next year the farm will be here but we won't."

Across several state lines the farmers had been replaced by steelworkers, but their problems remained much the same. In the early morning hour twelve of the first twenty people in the Salvation Army soup line in Pittsburgh were children, one a baby in her mother's arms. "People go to bed hungry in western Pennsylvania," we were told by Dr. Gordon McLeod, former state secretary of health. "We have a serious problem, a return to macaroni, bean soup and white bread." This is steel country where many residents are highly skilled production workers. More than 200 food pantries have sprung up in the Pittsburgh area, some affiliated with medical clinics that found more people in need of food than a doctor. Children's television personality Fred Rogers of Mr. Roger's Neighborhood broadcasts from Pittsburgh. "It makes you feel so bad," he said, "to hear about children without food."

Three doctors stared into a sea of faces at the town hall library in McDowell County, W. Virginia. A group of about seventy parents, equally divided between blacks and whites, listened as one of the doctors spoke: "How many of you find you cannot feed your children properly nowadays?" More than half the parents raised their hands. One asks these people how the national economic boom is affecting them and they stare at you. Some smile. McDowellCounty is poverty, pure and simple. Nothing indicates that life is improving. "How many of you think life will be better for your children, "another doctor asked the parents in the room. No hands went up.

"We see no end to the need for food around here, "Betty Drake stated. "Families come to our agency all the time saying they have nothing to feed their children. I see fourteen month-olds who are so small they look half their ages."

Despite growing hunger seen by local professionals, the town ended its summer feeding program for 200 children. U.S. Agriculture Department officials said the program violated some technical provisions of federal regulations. "We think their real goal is to close the program," charged agency worker Audrey Harriston. "A lot of people we know had hungry kids last summer."

The pastor of the First United Methodist church in the town of Welch reported an increasing number of parishioners going hungry. "It's hard to watch," he said. "Coal miners never before on assistance cry to get help. I didn't think things like this happened in America.

John McCone of the State AFL-CIO charged that federal policies permit employers to work people at an inadequate minimum wage, one so low that they remain in poverty while working full-time. "The whole business is a disgrace," Florette Angel of the State Youth Coalition told us. "A rape of the Statue of Liberty and of the middle class. Shelters and soup kitchens shouldn't be here in the first place, not in America.

The sheer size of California makes it significant, the nation's largest population, home to one of every ten Americans. From the high-tech economy of the Silicon Valley to the sunny communities of San Bernardino Valley, California projects itself as a land of opportunity, a vision of tomorrow. Behind the image lies a terrible truth. Some 3.6 million residents live in poverty, a million and a half of them children. Assembly line workers join agricultural laborers earning under $10,000 a year. Millions have no health insurance, living one paycheck from disaster. Poverty rose dramatically in the early part of this decade, some 65% among children in just four years. Along with this increase came other factors associated with low wages-- growing numbers of mothers who cannot get prenatal care, and for the first time in two decades, an increase in infant mortality in the state. Alice Callahan sees families with no food every day in her Los Angeles agency. "Some haven't eaten for two or three days. Children literally go through garbage cans."

Sister Rita Russo reports the same tragedy at her Seedling Food Center where she feeds 850 families a month. Two thirds of them are children. "Every day we find people who have not eaten for a couple of days," she observed. Some walk the sixty-three blocks from the downtown area to reach her facility.

Lloyd Grimes, a steelworker for twenty years, now lives on rice and government cheese. His wife is ill and his fifteen year-old daughter developed ulcers while the family was undergoing its adjustment to unemployment. "My life's a standstill," Lloyd said. "I feel degraded. Worthless". He is one of 400,000 blue collar workers in L.A. who were displaced by plant closings in recent years. Many now stand in the bread lines that have grown up across the city.

In the heart of Santa Clara County to the north lies San Jose, one of the more affluent cities in the nation. This former cannery community has been transformed into a center of the nation's high-tech industry. Microelectronics is the shining jewel of the Silicon Valley crown, but it is a crown filled with many lesser-shining stones. The workforce is comprised largely of young women who toil at low-wage jobs with few benefits. Mostly non-unionized, they seek out an existence in an area with one of the highest median incomes in the nation. Many of these workers and their families experience periodic hunger, and their children frequently eat donated food. Every month the Santa Clara food bank serves 35,000 people, about 85% of them households with children.

Religious leaders in the area call the plight of these workers the "underbelly" of the California economy, the smudge on the gloss of the high-tech miracle. It seems important to ponder the paradox of California, a state of wealth and promise where 2.3 million people-- one of every ten-- rely on emergency food charity each month.

Many Californians like to see their state as a trend-setter, a harbinger of the future. But not all the trends are good ones. Los Angeles has the largest homeless population in America, the country's largest food bank, and the largest program to keep babies from being born malnourished. In San Jose the state has some of the most disparate incomes in the U.S., where impoverished families live in the shadows of those who have among the highest incomes anywhere. California, the trend-setter, may be a window on a nation that has more and more jobs at lower and lower wages. The state may represent an emerging society of hard-working have-nots.

In the early days of the Reagan Administration, Office of Management and Budget Director, David Stockman vigorously advanced a "trickle--down" theory of economic development. Officials today point to economic trends as proof of their original theses. The President observes that econimic growth is (quote) "building a better life for our nation's families." Yet within the prosperity to which he alludes are troublesome trends. When government officials present aggregate data on the economy things look good for everyone. But when the numbers are disaggregated major sectors of the economy are lagging farther behind. When, for example, an employer hires two people part-time rather than one full-time, as a way to avoid paying health insurance and other benefits, the government tallies up two jobs where before there was only one. But behind the higher job count is a family whose income has dropped precipitously. The President's data do not reveal their suffering.

It is in this sense that we must peel away the veneer of prosperity to examine why hunger continues at epidemic proportions in the nation, even while the economy improves for some. Several factors explain the seeming paradox of prosperity and hunger. First, poverty is higher today than at any time from 1969 to 1980. While poverty has dipped and little in recent years, more Americans live in poverty than when the decade began. Over 13% of our people live in poverty throughout the year, and a full 25% live in poverty sometime during the year. But the official poverty level of $9,000 for a family of three does not depict the degree of suffering--fully 40% of all people in poverty have incomes which are less than half the official poverty line.

A popular explanation for high poverty is the American family structure, namely households headed by women. While research shows this to be a factor, it is not a principal cause of poverty. Since 1978, the period in which poverty has increased, it has in fact become slightly less "feminized". Nearly half the growth in poverty, in fact, has been among families headed by males or non-elderly couples. Thus, it is the economy and public policies to which we must look to understand the unusually high level of poverty in the nation.

A second factor that arises is the minimum wage. The current minimum of $3.35 an hour was last set under President Carter in 1981. Since that time inflation has grown by 32%, meaning that families with earnings at the minimum wage level have seen nearly a third of their incomes eaten away through no fault of their own. A rationale for the low minimum wage is that it offers jobs to youth entering the labor market, especially hard-pressed minority youngsters. Yet the facts depict this claim as a rationale for exploitation. About 70% of minimum-wage workers are adults, and over 80% of them are white.

A third factor of significance--one very troubling for our country-- is the dramatic shift to low-paying jobs that is taking place. By 1987, real wages in the nation were lower than for any year in the 1970's. The chief economist for the Joint Economic Committee of Congress noted that there has been a "total slippage of real wages in the work force." The number of people who work but remain in poverty has grown 50% since 1978. Of the 1.3 million jobs created during this Administration, over 8 million are at wages of less than $7,000 a year. Half of all jobs created during this decade, in fact, fail to keep families out of poverty. At the same time, the number of jobs paying over $14,000 annually has declined by nearly two million. The dramatic shift to low-wage jobs, in conjunction with the other factors I have mentioned, served to render larger segments of our population vulnerable to increasing economic disparities associated with hunger.

Another factor which helps us to understand the existence of hunger is the intentional weakening of federal programs designed to lift people out of poverty. These programs--housing, public assistance, food stamps, medicare and medicaid--are having less impact today than when the government began measuring their antipoverty effect back in 1979. In that year, these programs lifted from poverty some 20% of families with children who would have been poor based on their cash incomes alone. By 1986, only 12% of such families were lifted from poverty. The weakening of these programs resulted from high inflation in the early part of the decade and large cuts in federal food and assistance programs at the start of this Administration.

As a result of changes in federal programs, the economy, and tax policies providing relief to the rich but not proportionately to the poor, income disparity in the nation has grown during the decade. The gap in after-tax income reached its widest point since the federal government began keeping this statistic some four decades ago. The richest 20% of families now gets over 43% of the national income. The poorest 40% of the nation gets 15% of the income. The recorded income gap between rich and poor is $40 billion. This means that the richest fifth of the nation gets this much more than it would were its income at the same proportion as it was in 1980. This is $40 billion shifted from the rest of the population. This increase in income disparity took place not because the rich worked harder than the poor or because they were more cleaver. It happened because those in power believed in a philosophy which holds that you help everybody indirectly by helping the rich directly. This is what "trickle-down" economics means.

The only problem is that it has clearly failed to work. What are we to do about domestic hunger?

Where is America to go on this highly-important issue?

Hunger grows from economic disparities and inappropriate governmental policies which perpetuate poverty. This knowledge gave us ability to substantially reduce hunger during the 1960's and 1970's, joining other western democracies which have eliminated such misery in their midst. But we let it return and once again we face the challenge to end hunger.

Solutions advanced by some of our highest leaders are terribly myopic. They have suggested that private charity is the way for a modern nation to eliminate hunger. But soup kitchens and bread lines, despite their commendable efforts, face an ever growing tide of hungry families and still their efforts are not enough.

The Second Harvest Food Bank System across the country increased food distribution by 1000% in five years, but the 100 million pounds of food distributed constitute a drop in the bucket compared to the need. Much of what they have to give away is not desirable from a nutritional perspective. They frequently receive and distribute the dregs discarded by the food industry--contents high in salt, sugar, and fats. The hungry, particularly hungry children and old people, should not have to rely on a diet of syrups, sugared cereals, pickles and pigs feet. Private charity is not a solution to domestic hunger, and hand-outs do not represent good nutrition policy for a modern nation.

We can end hunger in America. But this will happen only when those of us who do not suffer become as concerned as those who do. Hunger in America will end-- if, and only if, we force our political leaders to reflect our decency in the public policies of our government.

Thank you very much.

Nutritional and Lifestyle Disorders in Blacks

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Living in America is from Rocky IV:

"Superhighways, coast to coast, Easy to get anywhere, On the Trans-continental, All alone, Just hide behind the wheel, How does it feel, When there's no Destination that's too far, And somewhere on the way, You must find out who you are, Living in America.."*

* From "Living In America"
Performed by James Brown
Written by Dan Hartman and Charlie Midnight
Video produced by Dan Hartman

Living in America -- The pace is fast; the road winds through diverse cultures, bridged by a common wish for the "good life". But the toll, in terms of health, can be high.

Finding out "who you are" is important; cultural backgrounds influence our lifestyles and health habits, and they can be significant in determining just how well we live in America.

The U.S. Department of Health and Human Services Black and Minority Health report says that four social characteristics are believed to be especially significant influences on minority health status in the U.S. today: demographic profiles, nutritional status and dietary practices, environmental and occupational exposures, and stress and coping patterns.

Because of the Darwinian theory of the "Survival of the Fittest," the descendants of the African slaves, brought to the New World have been able to produce outstanding athletes such as Pele, the soccer player, and now the heavyweight fighter Mike Tyson, who is expected to be another Joe Louis. Yet morbidity and mortality statistics are higher for Blacks than for any other ethnic group, due to the dual influence of environment and lifestyle. Black athletes may be examples of the "Survival of the Fittest". Physical labor, however, is disappearing from our jobs and lifestyles. Low income Blacks are using nicotine, alcohol and other drugs to, as Bertrand Russell once said, "Create the illusion that it's better to be alive than dead". The benefits of starting an exercise program are such that a program often induces people to subsequently begin other health practices, such as weight loss, diet changes, avoiding alcohol and stopping smoking.

A July, 1987 preliminary report from the National Institutes of Health Study on Centenarians revealed that the U.S. has 25,000 persons 100 years of age or older, and that by the year 2000 the number will increase to 100,000.

As a group, the centenarians are socially and economically diverse. Characteristics are similar to the population aged 85 and older. Centenarians are more likely to be women, more likely to be living with someone other than a spouse, and have low incomes. More than 75 percent of the women are widowed; more than 20 percent of the men 100 or older are still married, compared to 10 percent of the women. Centenarians are found at all levels of educational attainment, but women are more likely to have high school diplomas. One-half of all centenarians live in households with others or by themselves; most of the remainder are in nursing homes.

More Blacks than Whites are found among the oldest centenarians, 105 years of age or older. A higher proportion of men among Black people have lived to be 100 years and older than among Whites who have lived to this age. Blacks are less likely to be found in institutions, but are more likely to be poor.

Poor patients are less likely to have continuity of care with a particular health care provider. Many minorities rely on Medicaid and charity care for medical treatment because they have no other resources. Elderly minority people are less likely than Whites to supplement Medicare with additional private insurance. Continuity of care improves health outcomes and is greater when patients establish ongoing relationships with particular health care providers. It is also important in the treatment of such major killers of minorities as cancer, cardiovascular disease, and diabetes, which are chronic and which require repeated visits and extended treatment regimens.

Throughout this presentation, many references will be made to information from the U.S. Department of Health and Human Services "Report of the Secretary's Task Force on Black and Minority Health".

Formation of the Task Force on Black and Minority Health in April, 1984, was a response to the national paradox of phenomenal scientific achievement and steady improvement in overall health status, while at the same time persistent, significant health inequities exist for minority Americans. According to the Task Force's findings, efforts of monumental proportions are needed to bring the health of minorities to the level of all Americans.

Analysis of mortality data from 1979 to 1981 revealed that six causes of death together account for more than 80 percent of the mortality observed among Blacks and minority groups in excess of that in the White population. They are (in alphabetical order):

- Cancer
- Cardiovascular disease and stroke
- Chemical dependency, measured by deaths due to cirrhosis
- Diabetes
- Homicide and accidents (unintentional injuries)
- Infant Mortality

Prevalence of arthritis, digestive diseases, dental problems and respiratory disease among minorities is poorly documented and data are virtually unavailable.

Some factors which contribute to minority health status are not disease-specific but have a bearing on the overall health needs of each minority group. The Task Force reviewed such factors as demographic data characterizing the four minority groups, minority needs in health education, health professionals, and health care services and financing.

Acknowledging that factors responsible for health disparity are complex and defy simplistic solution, the Task Force noted that health status is influenced by the interaction of physiological, cultural, psychological, and societal factors which are poorly understood for the general population, and less so for minorities.

Although the shared characteristics of economic disadvantage among minorities suggest common approaches for achieving improved health, diversity within and among minorities necessitates activities, programs, and data collection tailored to each group's needs. All sectors must work to rectify the under-representation of minorities as beneficiaries of the non-minority health care establishment.

Improvements are needed in the following areas:

- Health information and education
- Delivering and financing health services
- Health professions' development
- Cooperative efforts with the non-Federal sector
- Data Development
- Research agenda

The Task Force noted the "stark reality of the continuing, significant differences between the health of racial/ethnic minorities in the U.S. and the general population and proposes recommendations for DHHS and others in the public health community to address these problems". Here are the Task Force's recommendations for achieving significant improvements in the health of minority groups by the year 2000:

Health Information Education:

Recommendation 1: An Outreach Campaign -- The Department should launch an outreach campaign to disseminate health information and education materials and program strategies specifically designed for minority sectors of the population, including Blacks, Hispanics, Asian/Pacific Islanders, and Native Americans. The campaign should strengthen and expand present departmental health promotion efforts emphasizing preventive aspects of those public health areas identified by the Task Force which have the greatest impact on death and disability among minority populations: cancer, cardiovascular disease, chemical dependency, diabetes, homicide, suicide and unintentional injuries, and infant mortality.

Recommendation 2: Patient Education -- The Department should ensure that its materials, programs and technical assistance for patient education are responsive to minority population needs, especially those provided in specific health care service settings such as medical, community-based, school and worksite locations. Emphasis should be given to those public health topics that have the greatest impact on death and disability in minority populations. Concomitantly, attention should be given by the Nation's schools and universities to training health care providers and educators to be sensitive to minority cultural and language needs.

Delivery and Financing of Health Services:

Recommendation 3: Delivery and financing of Health Services -- The Department should continue to investigate, develop and implement innovative models for delivery and financing of health services, based on current departmental authorizations. The major objectives of the models should be to increase flexibility of health care delivery, facilitate access to services by minority populations, improve efficiency of service and payment systems, and modify services to be more culturally acceptable.

Health Professions Development:

Recommendation 4: Developing Strategies Outside the Federal Sector -- The Department should initiate discussion with minority and non-minority health professional organizations, academic institutions, State governments and health departments, and other entities from the public and private sectors, to develop strategies to improve the availability and accessibility of health professionals to minority communities.

Recommendation 5: Developing Strategies Within the Federal Sector -- The Department should conduct both intra- and interdepartmental reviews identify and provide for collaboration between the various activities currently being supported within the Department and other elements of the Executive Branch, respectively. The reviews should focus on programs that have impact on the actual or potential availability of health professionals to minority communities.

Cooperative Efforts with the Non-Federal Sector:

Recommendation 6: Build the capacity of the Non-Federal sector to address Minority Health Problems – DHHS should increase its involvement with State, local, and community agencies and organizations to encourage efforts specifically oriented toward meeting minority health needs. Recognizing that communities have unique insights into their own health problems, DHHS should undertake activities to increase awareness by minority communities of the Federal ability to provide an overview of health problems and to provide technical assistance.

Data Development:

Recommendation 7: Improving and Fully Using Available Sources of Data -- DHHS should undertake activities, which would improve existing sources of health data, such as enhancing cooperative efforts with the States in recording vital statistics, incorporating specific racial/ethnic identifiers in data bases, and oversampling selected minorities in national surveys. Furthermore, DHHS should support innovative uses of currently available data consistent with the Privacy Act and confidentiality constraints. Analyses such as cross-comparisons from different data sets and specialized studies should be encouraged because they can contribute to understanding the health status and needs of minority populations.

Research Agenda:

Recommendation 8: Research Agenda – The Department should adopt and foster research agenda to investigate factors affecting minority health and should incorporate appropriate research activities on minority health into ongoing research programs consistent with the referral guidelines of each DHHS agency. The Task Force considers the following areas to be of major importance for research:

- Risk factor identification
- Risk factor prevalence
- Health education interventions
- Preventive services interventions
- Treatment services
- Sociocultural factors and health outcomes

In a letter to The Honorable Margaret M. Heckler, then Secretary of DHHS, which introduces the Task Force report, Thomas E. Malone, Ph.D., Task Force chairman, said:

"...If these recommendations can be implemented with the same spirit as their genesis, we will advance as a Department and as a Nation toward the health status of minority Americans today, with the assurance that we will all be healthier Americans tomorrow."

Ms. Heckler's letter in the report's introduction noted that the disparity in the burden of death and illness experienced by Blacks and other minority Americans has existed ever since accurate federal record keeping began more than a generation ago. She said that the Task Force's assignment was to "unravel the complex picture provided by our data and experience." She concluded with hope that the report can and should "mark the beginning of the end of the health disparity that has for so long, cast a shadow on the otherwise splendid American track record of ever improving health".

Having summarized this report, I would now like to turn to some aspects of:

A. HYPERTENSION -- a major contributor to the excess morbidity and mortality

1. Nearly 1 in 6 Americans have it and only a few are treated because symptoms go undetected.
2. Estimates are that 1 in 3 Black Americans have it, even very young Black women are more susceptible than white women and it is usually more serious in Black women. Blacks have five times the malignant variety and 10 times the renal complications. Stroke differences in Blacks and Whites.

3. The sodium factor in ethnic diets.
4. Sodium content in common foods and how to cut down easily.
5. Vegetarian diets can reverse atherosclerosis.

B. HYPERTENSION, CARDIOVASCULAR DISEASE, OBESITY, DIABETES AND EXERCISE

1. Black Americans' Lifestyles

a. Mainstream Blacks have less poverty but greater risks of cardiovascular disease from increased blood cholesterol, smoking, and decreased physical activity. Male black physicians have a heart attack rate 10 times greater than their white counterparts, according to a preliminary study by Dr. John Thomas of Meharry Medical College.

b. Black women are more likely to be obese than black men who usually do more physical labor.

C. VIOLENCE PREVENTION

1. Educational programs which teach non-violent means of dealing with problems and situations.

D. SPIRITUALITY -- a protective factor

Faith is one of the most important elements in preventive medicine. Preventive medicine needs to take a total approach to the individual which is philosophical; illnesses that have a spiritual component cannot totally be explained in terms of hard science and Newtonian physics. There is another force in the universe that we feel but do not completely understand.

1. The Faith Factor: Wanting to get up in the morning
2. Alcohol and Drug Abuse, 12 Step AA Plan
3. Obese Persons' Addition to Food
4. Health Benefits and Spiritual Focus of Fasting: M. K. Gandhi

Another significant influence on the health status of all Americans, minority or not, is that despite the trend toward healthier lifestyles, we still have the tendency to focus more intensely on curing disease than we do on preventing it. Also, health is still a matter of wealth; the fitness movement hasn't trickled down to lower income and minority people, who are less likely to have access to adequate medical care whether it's for sickness or wellness.

It has been estimated that at least 70 percent of patients treated by primary care physicians are neither sick nor well, don't need drugs to cure their ailments, and really need rest or relief from stress.

In a June, 1985 article in The Futurist, "No Limits to Health", author/publisher Robert Rodale says, "People can get more health not by getting more medicine or medicalized care, but by doing and thinking the things that will generate more health from within themselves". Emphasizing the need to transform our health philosophy, Rodale noted rising costs of medical and hospital care, and called for a change in the way government, schools and universities, businesses, and other institutions allocate resources in the hope of creating health. He said that treatment gets 96-98 percent of all health resources in the U.S.

I agree with those who emphasize the importance of preventing disease. Not only does prevention add to the quality of life, but ultimately, it is the best way to cut health care costs. We in the West spend far too much of our time and money on curing, when we ought to be preventing diseases, most of which are preventable. We're spending our time and money trying to pick up the pieces instead of trying to prevent the casualties.

The half-way progress report given by Dr. Michael McGinnis, deputy Assistant Secretary for Health, to the media in 1986, noted that progress has been made in some areas: Heart attack deaths were reduced by 34 percent and stroke deaths by 48 percent from 1972 to 1984. Mortality rates at each of the major life stages have been improved, half-way or better, since the goals were set. For example, by 1985, infant mortality rates had been reduced by 25 percent; the goal was 35 percent. But efforts to combat teen pregnancy, drug abuse and smoking among young women must be strengthened, Dr. McGinnis said.

While the 30 percent reduction in adult smoking in the last 20 years can be called a spectacular result, the increase in smoking among young women ages 20-24 is a serious concern. The 13 percent increase in cocaine abuse from 1984 to 1985 is also a very alarming situation. The well-publicized 1986 War Against Drugs fostered more widespread use of testing and beefed up law enforcement efforts to interrupt the illegal supply lines, but not much money or attention was allocated for rehabilitation and research to find out reasons for drug abuse.

The problem of teen pregnancy with its far-reaching social and economic effects on our future is being called an epidemic with just cause. Marion Wright Edelman, president of the Children's Defense Fund, reported in March, 1986 that each day 3,000 American girls get pregnant; 1300 give birth, and that teenagers produce enough babies annually to fill the cities of Denver and Atlanta. The National Academy of Sciences has called for more availability of contraceptives and family planning services to adolescents. Speaking for the Academy, Dr. Daniel Fellerman called for a greater public awareness and social consciousness from all sources -- family, schools, television, and others.

In the grand scheme of things -- health goals, socio-economic improvement plans, enhancement of the quality of life by various programs -- minority groups, generally thought to be the ones receiving most of the "welfare" aid, don't show a "most improved" rating on the statistical report card. Minority longevity and infant mortality rates do not equal those of whites.

Living in America, for those who belong to most minority groups, means not living as well or as long as non-minorities because of inadequate or non-existant health care and health care awareness. That's what this paper is about: Awareness. All people can enter into partnerships with health care professionals to work toward their own health improvement and maintenance. They can also learn to live healthy lifestyles, use PREVENTION, and make improvements on their own.

When more than 200 leading biological scientists convened in London early in 1987 to discuss medicine in the year 2000, they predicted that prevention will have a greater impact on cancer, heart disease and other diseases. They were optimistic about the new genetic revolution in biotechnology which is expected to result in new vaccines for prevention. They predicted that preventive methods will be more beneficial than treatment or early diagnosis and that changes in lifestyle and nutrition will also have a major impact on health by the year 2000. Dr. Denis Parsons Burkitt, whose opinions are well-known, participated in the London conference. In a media interview, he said that up to now, medical treatment has had little impact on reducing disease and mortality, and that prevention is the only way to deal with the health problems which are due mainly, to the drastic changes in our diets and lifestyles made in the past 150-200 years.

The National Cancer Institute has said in its Cancer Control Objectives for the Nation, that the number of deaths due to cancer could still be cut in half by the year 2000 by increasing the use of current knowledge about prevention, screening and treatment. The report notes that smoking impacts on 30 percent of cancer mortality and is directly responsible for 30 percent of the 450,000 deaths due to cancer annually. If the cancer rate due to smoking could be reduced to 15 percent or less, the overall death rate due to cancer could be reduced by 18 percent.

Although the effect of diet on cancer is not clear cut, different studies have indicated that 10 to 70 percent of cancer mortality is related to diet. A study by two British epidemiologists suggests that about 35 percent of cancer mortality is directly related to diet. If Americans would reduce their fat intakes to no more than 30 percent of the total calories, and increase their fiber intake to 20 to 30 grams per day, it is believed that overall total mortality rate due to cancer would drop by about 8 percent.

The effectiveness of cancer screenings is a good example of how current knowledge could be used more frequently and by more people to reduce cancer mortality. Breast cancer screening with mammography could prevent 30 percent of the breast cancer deaths, if women over 50 would be screened annually with a physical examination as well as mammography. Approximately 50 percent of women get annual pap smears to test for cervical cancer, but if the number could be raised to 80 to 90 percent, the mortality rate for this cancer would be reduced. And, if all women are screened annually for breast and cervical cancers as often as is recommended by the Cancer Institute, the overall cancer mortality rate would be reduced by about 3 percent.

Surveillance of Epidemiology and End Results (SEER) has shown that all cancer patients are not getting the state of the art treatment which impacts the survival rates. For example, the survival rate for breast cancer is about 66 percent in post-menopausal women. The American Cancer Institute's panel of experts believes that the rate could be as high as 72 percent just using information that is known today. The survival rate for colon cancer is about 41 percent, but it could be 51 percent; the rate for rectal cancer is 38 percent but it could be 59 percent.

The American Cancer Institute says that if improved survival rates for cancer at all sites are translated to mortality reduction, the result would be a 10 percent reduction in the overall cancer rate. Successful reduction of the overall cancer mortality depends upon the cooperation of the public, employers, the health professions, news media and the government.

Preventive medicine is too often under-emphasized in the Black community. A recent symposium sponsored by the Black Medical Students' Organization from Johns Hopkins University set the following goals:

1. To raise Black community's awareness level about the importance of good health.
2. To emphasize to health care givers and potential health care givers the importance of preventive medicine.
3. To suggest simple, inexpensive measures which can lead to a healthier lifestyle among residents of the Black community.
4. To emphasize that diseases for which Blacks are considered at high risk can be prevented, or lessened in severity as a result of preventive measures.

The students were concerned about such topics as nutrition on a limited budget, understanding and avoiding occupational and environmental hazards, stress and mental health factors as they relate to physical well-being, and the information barrier between the health care giver and the community.

Dr. Holly Flack, of Howard University, and Dr. Edward Pelegrino, Director of the Kennedy Ethics Institute of Georgetown University, say that Blacks conceptualize health problems differently from the majority of the population. A lack of information as well as lack of financial and medical resources has fostered a sense of prolonged helplessness in the Black community about health issues -- a situation that needs to be overcome by establishing a panel of people trained to deal with health from the Black perspective.

Dr. Flack says insufficient money has gone into research projects dealing with health issues affecting minorities, and that Black institutions have been strapped for cash so that they can't do research; they lack the laboratories or the personnel needed to run meaningful research.

Dr. Pelegrino feels that Blacks still have to turn to Black institutions for research on Black health problems. One of the problems is that Blacks have traditionally been teachers instead of researchers, or feel obliged to fill the ranks of the health and medical professions with Blacks. Research has been considered a luxury, and teaching has always been a priority.

Dr. Flack says there needs to be a sense of community and a concern about Black issues that affect the community as a whole, rather than a focus on individual needs, such as is more prevalent among the majority groups. While the

Black consumer is becoming more aware of the need for health care, more attention is needed to focus on delivery of accurate information about Black patients in a way that is consistent with minority cultural considerations.

There is also a need to deliver accurate information about Black and other minority patients in a way that is statistically precise. For example, according to data from the National Hospital Discharge Survey, mortality rates in the U.S. from coronary artery disease are higher among blacks than whites at younger ages, with a crossover to lower rates above the age of 70. The factors which determine this crossover of age-specific death rates have not been adequately explained. It is known that coronary artery disease has an earlier onset in the black population.

Analysis by a research team at Cook County Hospital in Chicago, of data from the National Hospital Discharge Survey for 1973-1984 showed age-related differences in case-fatality rates between whites and non-whites. In-hospital fatality rates were 10 to 70 percent higher for each of the 10-year age groups for non-whites up to age 70, when a crossover occurred. The median age at death from myocardial infarction was approximately 5 years younger in non-whites compared with whites. National estimates of hospitalization rates for myocardial infarction suggest that non-whites receive less health care for coronary artery disease than whites relative to recorded fatal events. Data from several community studies have shown that in-hospital death rates from coronary artery disease are lower among blacks than whites, accompanied by an excess of out-of-hospital deaths. It has been suggested that the higher rates reported among blacks in the vital records result primarily from misclassification of these medically unattended deaths.

The Cook County Hospital team concluded that age-specific trends in case-fatality support the hypothesis that a cohort selection effect partly determines the black/white differentials in coronary artery disease. Also, that the relative susceptibility of the Black and White populations is not appropriately estimated by age-adjusted rates, but should be examined on an age-specific basis within the framework of selection effects on a cohort.

It seems appropriate to conclude that the 1986 top honor in fuzzy thinking, according to judges at Lake Superior State College, Michigan, which annually lists pointless, redundant and inappropriate phrases and linguistic atrocities, went to a physician instead of a politician for the first time in 11 years. The "ultimte in stinko use of language" was a physician's note that "the patient did not fulfill his wellness potential", which according to the judges, "not only obscures the fact that the patient died, but places the blame squarely on him (the patient)". Other "linguistic atrocities" committed by physicians listed by the judges were referring to patients as "clients" and the practice of sending babies to "neonatal units" instead of to nurseries.

The perception of the public is that they are "clients" and that the medical profession is more interested in money than in patient care. However, nearly everyone believes that medical care will ultimately become so expensive and that self-care will become a necessity, in order to keep people from going into the hospital and risking the loss of their assets. And, the best self-care is prevention through adoption of healthy lifestyles.

Food and Nutrition Issues of American Indians and Alaska Natives

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According to the 1980 census, there were 1,418,195 American Indians, Eskimos, and Aleuts in the United States. Of this number, 32 percent lived on designated reservations and historical areas of Oklahoma, 5 percent lived in Alaska Native villages and tribal trust land, and 63 percent lived throughout the remainder of the United States (1).

American Indians and Alaska Natives represent numerous tribes, with diversities in religion, language, traditions, beliefs, attitudes, and values. As of April 1984, there were 291 Indian tribes and 197 Alaska villages designated as federally recognized (2). The federal government's responsibility for these recognized tribes has origins in treaties signed between the tribes and the U.S. government, and further established through congressional mandates.

Demographics

The 1980 census data indicate that the American Indian and Alaska Native population is younger than the U.S. All Races population; they have a higher birth rate; and they lag behind the U.S. All Races population in respect to income, employment, and education (1,3). As shown in Table 1, 32 percent of the American Indian and Alaska Native population was under 15 years of age, as compared to 23 percent for the U.S. All Races population. In addition, 5 percent of the Indian population was over 65 years of age. Eleven percent of the U.S. All Races population was over 65 years old.

TABLE 1: Percent Distribution of the Total American Indian and Alaska Native Population for Reservation States and U.S. All Races by Age, 1980 Census Data

Age	American Indian/ Alaska Native*	U.S. All Races*
Under 5 years	10.8	7.2
5-9 years	10.5	7.4
10-19 years	23.3	17.4
20-29 years	19.1	18.0
30-39 years	13.2	13.9
40-49 years	8.8	10.0
50-59 years	6.7	10.3
60-69 years	4.4	8.3
70-79 years	2.4	5.1
80 years and over	0.9	2.3
Median Age	22.6 years	30.0 years

*Percentages may not add to 100 due to rounding

For the years 1982-1984, the Indian and Alaska Native birth rate was 28.8 live births per 1,000 population. This was nearly twice the U.S. All Races rate for 1983 of 15.5, and almost one third higher than the U.S. other than White rate of 21.3 (4).

Table 2 illustrates various social and economic characteristics. In addition to these characteristics, of the 278 Indian Reservations surveyed by the Census Bureau in a supplementary questionnaire, 56 percent (81,273) of the housing units had no telephone; 16 percent of the housing units lacked electricity; 17 percent lacked refrigeration; and 21 percent lacked indoor toilets. The Hopi and Navajo Reservations in Arizona and New Mexico had the largest population without electricity and refrigeration (3).

TABLE 2: Social and Economic Characteristics of the American Indian and Alaska Native Population and the U.S. All Races Population, 1980 Census Data

Characteristics	**American Indian, Eskimos & Aleuts**	**U.S. All Races**
Mean Age	22.6	40.0
Percent Female	50.7	51.4
Percent Male	49.3	48.6
Average No. Persons Per Family	4.6	3.8
Median Family Income (4)	3,700.	19,900.
Average Family Income	16,500.	23,100.
Per Capita Income	3,600.	7,300.
% Persons below poverty level	28.2	12.4
High School Graduates, %	55.3	66.5
College Grads, %	7.4	12.2

Health and Nutritional Status

Diseases and mortality among American Indians and Alaska Natives today are different from those prevalent two to three generations ago. Since 1955, the health status has greatly improved, as shown in Tables 3 and 4. There has been 82 percent decrease in infant death rate; 89 percent decrease in the maternal mortality rate; 96 percent decrease in pneumonia and influenza; and 93 percent decrease in tuberculosis.

TABLE 3: Vital Statistics for the American Indian and Alaska Native Population, Calendar Years 1954-56 and Calendar Years 1981-1983 (4,5).

Vital Statistics	**Calendar 1954-195**	**Calendar 1981-1983**	**% Decrease**
Infant Death Rate (per 1,000 live births)	62.7	11.0	82
Neonatal Death Rate (per 1,000 live births)	23.1	5.0	78
Maternal Death Rate (per 100,000 live births)	82.6	8.9	89
Pneumonia and influenza (per 100,000 pop.)	89.8	6.2	82
Tuberculosis (per 100,000 pop.)	55.1	2.3	96

TABLE 4: Age-adjusted Mortality Rates, Deaths per 100,000 Population, for Selected Causes, 1955 and 1982

Diseases	**Indian Alaska Native**		**U.S. All Races**	
	1955	1982	1955	1982
Heart Disease	135.8	176.2	190.5	238.3
Diabetes	17.0	19.9	13.0	9.6
Tuberculosis	57.9	2.0	8.4	0.6
Pneumonia and Influenza	89.8	17.4	25.4	10.9
Alcoholism	56.6*	35.8	7.7*	6.4
Gastrointestional Disease	15.4	3.1	3.6	2.7

Chronic diseases are now the prevalent health concerns. Hypertensive disease and diabetes mellitus, primarily Type 2, were among the leading reasons for outpatient visits in the Indian Health Service facilities in fiscal year 1986. Heart diseases are the leading causes of death (4). Included among the nutrition related health concerns of American Indians and Alaska Natives are obesity, diabetes, hypertension, and alcoholism.

Obesity

Obesity is increasingly becoming a significant problem in the Indian and Alaska Native populations, especially as it relates to diabetes and hypertension. We have no national data on the prevalence of obesity, but several studies on individual tribal populations have been reported in the literature.

Obesity is prevalent in all southwest desert tribes surveyed by Sievers and Cannon (6,7). Body weight exceeding the ideal by more than 25 percent was seen in 39 percent of the men and 65 percent of the women. In a survey on the Navajo Reservation in 1983, Wolfe (8) found 63 percent of the women sampled to have weights greater than 120 percent of their ideal weight (8).

In a 1980 study of Seminole Indians in Florida, Joos (9) reported that 60 percent of the patients age 20 years and older, seen at the local Indian clinic, were obese. Similarly, in a recent study of 1,857 American Indians representing 10 tribes in Oklahoma, Lee and co-workers(10) found 75 percent of the subjects to be obese. On the average, they weighed 145 percent of ideal.

Diabetes Mellitus

Non-insulin dependent diabetes mellitus (NIDDM or Type 2) is more common in American Indians than in non-Indians. Diabetes is the seventh leading cause of death among the Indian population served by the Indian Health Service (4 , 11).

As shown in Table 5, the 1984 age adjusted diabetes death rate of 20.6 for Indians was 2.2 times the U.S. All Races Rate of 9.5, and 1.2 times the U.S. Other than White rate of 17.4 (4).

Diabetes was uncommon in American Indian tribes prior to 1940 (12). Today, tribes with reported prevalence rates of 20 percent or greater among adults 35 years old or older include: Pima, Upland Yuman, Maricopa, San Carols Apache, Cocopah, Zuni, Paiute, Seminole (both Florida and Oklahoma), Cherokee (both North Carolina and Oklahoma), Pawnee, Cousshatta, Seneca, Cheyenne-Arapaho, Shawnee, and Creek (13).

TABLE 5: Age-adjusted Diabetes Mellitus Mortality Rates per 100,000 Population for Indians and Alaska Natives in Reservation States and for the U.S. All Races and Other than White Population (4).

Calendar Year	Indian/ Alaska Natives	U.S. All Races	U.S. Other Than White
1984	20.6	9.5	17.4
1983	30.5	9.9	17.8
1980	22.6	10.1	18.8
1975	20.8	11.6	21.7
1970	27.1	14.1	25.2
1965	25.4	13.3	23.6
1960	20.3	13.6	21.6
1955	17.0	13.0	16.5

Genetic predisposition, longevity, and obesity have been identified as the main predictors for the development of diabetes. Recent changes in food behavior are implicated in the development of obesity in the Indian population. The increase in obesity is concurrent with the increase in diabetes.

Hypertension

Hypertensive disorders are both directly and indirectly related to mortality. In fiscal year 1986, hypertensive disease was the fifth leading cause of outpatient visits to Indian Health Service facilities (4). Both dietary factors and weight are implicated in the development of hypertension.

As with obesity and diabetes, the prevalence of hypertension is increasing in the American Indian and Alaska Native population. In the late 1970's, a blood pressure survey was conducted on 640 Navajo Indians (14). Of the normal weight subjects, age 20 and older, 18 percent of the Navajo men and 5 percent of Navajo women had high blood pressure. Of the overweight subjects, 43 percent of the men and 15 percent of the women had high blood pressure. Similar results were found in urban American Indians in Minneapolis (15).

In a recent report, the hypertension rate of Penobscot Indians of Maine was reported to be nearly the same as their non-Indian neighbors. However, their awareness of hypertension was significantly less (16).

Alcoholism

There has been a long standing recognition that alcohol abuse is a major problem among American Indians and Alaska Natives. Table 6 shows the age-adjusted alcoholism death rates. In 1984, the Indian death rate of 30.0 was 4.8 times the U.S. All Races rate of 6.2 (4).

TABLE 6: Age-adjusted Alcoholism Mortality Rates per 100,000 Population for Indians and Alaska Natives in Reservation States and for U.S. All Races (4).

Calendar Year	Indian and Alaska Natives	U.S. All Races
1984	30.0	6.2
1983	28.9	6.1
1980	41.3	7.5
1975	62.2	8.6
1970	56.2	8.1

Note: For 1970 and 75, includes deaths due to alcoholism, alcoholic psychoses, and cirrhosis of the liver with mention of alcoholism. For 1980 and after, includes deaths due to alcohol dependence syndrome, alcoholic psychoses and chronic liver disease and cirrhosis, specified as alcoholic.

Malnutrition is associated with alcoholism. In addition, chronic alcohol use can result in both marginal and chronic nutritional inadequacies.

Nutritional Status

The nutritional status of American Indians and Alaska Natives is largely unknown. Food and nutrition intake studies reported in the literature indicate that the nutritional adequacy of diets varies from tribe to tribe because of differing sociocultural and economic factors, as well as food availability and food preferences (17).

A few nutrition status surveys have been conducted in specific geographical areas. Nutrition studies were conducted on two cohorts of White Mountain Apache preschool children in 1969 and 1976 (18). In 1969, 41 percent of the children had low intakes of energy as compared to 16 percent in 1976. Similarly, in 1969, 41 percent had low calcium intakes, 36 percent had low vitamin C intakes, and 62 percent had low intakes of iron. In 1976, low intakes of calcium were 23 percent, 17 percent for vitamin C, and 38 percent for iron.

In 1974, the diets of Hopi Indian women were found to be low in calcium, iron, zinc, vitamins A, C, B-6, and folacin (19). Also in the early 1970's, the diets of Cocopah Indians were found to be low in calcium, iron, vitamins A, C, B-6 and pantothenic acid (20).

In 1978, Knapp and Panruk (21), conducted a dietary survey in Southwest Alaska, and compared their results to a dietary survey conducted in the same region by Heller and Scott from 1956 to 1961 (22). In general, caloric intake had declined; protein, iron, and B-vitamin intake had decreased, and the intake of low nutrient density carbohydrates had increased.

The dietary intake of Cherokee Indian adolescents in North Carolina was assessed as part of a study to compare food intake practices between fat and lean Indian teenagers (23). Diets were found to be low in calcium and iron.

The nutritional status of the American Indian and Alaska Native population is largely unknown because this population is essentially excluded from all major national nutrition surveys. The first Health and Nutrition Examination Survey (HANES I) was conducted between 1971 and 1973 (24). The purpose of the survey was to establish a continuing national nutrition surveillance system by measuring the nutritional status of the population and monitoring changes in status over time. Additionally, it was to help identify groups at nutritional risk. However, due to operational difficulties, "all people residing upon any of the reservation lands set aside for the use of American Indians would be excluded from the target population of HANES" (24). By the time

HANES II was conducted from 1976 to 1980, Hawaii, Alaska, and Indian reservations were added to the sampling areas (25). However, to my knowledge, data reported from HANES II are for Whites, Blacks, and Others. Indian and Alaska Native data are not broken out of the "other" classification. HANES III data collection is to begin this year and I anticipate the Indian and Alaska Native population will once again be "lost" in the data analysis.

Surveillance data relating to nutritional status in low-income populations are collected by the Centers for Disease Control (CDC) for two high-risk groups, children and pregnant women. Since 1974, CDC Pediatric Nutrition Surveillance System has collected data on children who attend public health clinics serving predominantly low income populations. In 1979, the CDC Pregnancy Nutrition Surveillance System was implemented. In interpreting the results from these surveys, CDC cautions that the number of children categorized as Native American was small, thus the data should be interpreted cautiously (26).

Access to food is an issue when looking at a population's nutritional status. Since at least 28 percent of the American Indian and Alaska Native population falls below the federal poverty level, I would like to discuss federal food assistance programs available to this population.

Food Assistance Programs

The government's involvement in providing food to Indians dates back to the beginning of the Reservation period. In 1834, Congress authorized the President to grant such rations as could be spared from Army provisions to Indians visiting military posts or Indian Agencies at the frontier. Most of the treaties of the 1860's provided for the issuance of rations. Rations were to substitute for hunting grounds given up by the tribes when put on reservations. Rations were the means to keep potentially hostile Indians away from Whites until alternative economic systems could be developed (27).

Today, varieties of food assistance programs are available to American Indians and Alaska Natives. Not all programs are operating at each Indian community, and some of the communities have none of the programs in operation.

In September, 1983, 127,699 Indian households participated in the U.S. Department of Agriculture's Family Food distribution Program on Indian Reservations, and an additional 107,198 households participated in the Food Stamp Program (28). These two programs contribute significantly to the American Indian diet. Wolfe (8) calculated that 40 to 50 percent of the intakes of most nutrients in Navajo diets surveyed were contributed by commodity foods. For example, the major sources of calcium in the Navajo diet came from bread because of the widespread practice of adding commodity nonfat dry milk to bread dough for tortillas and fry bread.

The Food Stamp Act of 1977 authorized the Family Food Distribution Program on Indian Reservations and required the Secretary of Agriculture to "improve the variety and quantity of commodities supplied to Indians in order to provide them an opportunity to obtain a more nutritious diet" (29). As a result, the current food package provides approximately 60 different foods, including canned meats and fish, cheese, butter, egg mix, peanuts and peanut butter, evaporated and dry milk, dry beans, canned fruit, fruit juices, canned vegetables, rice oats, cornmeal, pasta, farina, flour, corn syrup, honey, and other surplus commodities when they are available.

In 1982, 84 Indian tribal organizations provided meals for their elders under the provisions of Title VI of the Older Americans Act. More than one million meals were provided to nearly 10,000 older Indians (30).

Indian tribes are eligible to manage and administer the U.S. Department of Agriculture Supplemental Food Program for Women, Infants, and Children, known as WIC. In September 1986, 41,271 American Indian women, infants, and children were enrolled in one of the 33 Independent Indian State WIC Agencies (31). Additional Indians received supplemental foods from WIC programs administered through state and local health agencies.

Although many Indians participate in the food assistance programs, it is not known the extent to which the programs are meeting the population's food needs; the number who are eligible for the program but who are not participating, and the number who are not eligible for the current programs, but who are in need of some type of food assistance.

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*The opinions expressed in this paper are those of the author and do not necessarily reflect those of the Indian Health Service.

Overview of Nutritional Health of Minorities: Discussion

Anonymous:

I appreciated a lot of what Dr. Brown said about the politics of hunger. We see that a lot, and many of the programs that are developed to correct the problem don't do much good because no one has thought about the delivery of services in the field where there are many operational constraints.

Carter, J. P.

Larry, I noticed my name in one of the slides shown when you were talking about the romance with American Indians. I would like to say that the data contained in that paper were turned over to Senator McGovern and you might recall that it was through McGovern's efforts that the Senate Select Committee on Nutrition was established. The latter produced some changes in the Navajo reservation in terms of turning the management of the food programs over to the tribal counsel. Thus, a study that appeared to be romance with the American Indian had some definite political agenda.

Darby, W. J., Jr.

I wonder if Dr. Jackson has any information on the training of health care personnel, including physicians, who are of American Indian origin?

Jackson, Yvonne M.

In Indian Health Service, specifically in nutrition and dietetics, we have approximately a staff of 95, and of these 16 are American Indians. Right now we are funding 8 scholarships for undergraduate students. Public law 94437 allows for funding of scholarships for health professionals who are American Indians and Alaska natives. Quite often the number of applications falls very far short of the available scholarships.

Margen, S.

I really wish to thank Larry Brown for leading the way in an area where some of us, particularly those in public health nutrition, should be in the forefront. Larry's work on hunger in America has been subjected to a great deal of criticism particularly from the USDA and other governmental agencies. Their argument is that no one has been able to "substantiate the numbers" often quoted in terms of dimensions of hunger in America. Larry equates hunger with poverty. The same approach is often used by economists, and there is indeed very strong correlation between poverty and caloric intake of any population. I do, however, think that what has been the fault of nutritionists is that we have not attempted to differentiate hunger from malnutrition. They are two different problems. The end of hunger may be malnutrition but it doesn't have to be and it is very difficult to work with a concept which so far defies objective measurement.

The other important thing which Larry did not allude to sufficiently is that even when our so called safety nets are in place, for instance our food stamps, they are really not adequate to take care of the problems.

Kinnard, M.

In the current climate of budget deficit reduction in the country, I wish to ask Dr. Brown what he realistically thinks that the federal government can do and will do to reduce hunger and poverty in the country?

Brown, J. L.

The writer Jonathan Colzol, a major critic of public education in the country, said that one of the problems with education in this country is that we teach our young people that the great moments in history occurred in the past or will occur in the future, and we don't help them capture and see the acute critical issues that face us today. We are living through one of the most significant times in our history right now when we are confronted with a budget deficit of the proportion that we face and yet we have more money than we have ever had in our history. We are being faced with a challenge of deciding what America is, what kind of people we are, where we are headed and what is our moral bottom line? Your question is an excellent one but I want to say that it also raises a bogus issue.

America is not a poor third world nation that has to make choices between equally important basic priorities. Every industrialized democracy in the world has basically eliminated the degrees of hunger and deprivation that we have in our country today. We have more money than all of them or some of them put together. We don't have a resource problem in this country. We have a priority problem often portrayed as a resource problem, and it is very imperative during this time that professionals, nutritionists, physicians, nurses and others speak out and be a part of the debate.

Kinnard, M.

Dr. Jackson referred earlier in her presentation to the high prevalence of diabetes in the American Indians and Alaska natives. Is periodontal disease rampant in these groups?

Jackson, Yvonne M.

We are in the process of looking at that problem. I will also mention that these communities have a major problem with nursing bottle caries.

Adebonojo, F. O.

If indeed diabetes is prevalent in the American Indian population, then should one look at the larger birth weights of the babies as something positive or as something to be concerned about?

Jackson, Yvonne M.

The Indian Health Service just had a conference last month and that was one of the issues brought up for discussion. Should we be proud of the low prevalence of low birth weight babies when we have such a high rate of diabetes?

Winick, M.

I think you can be very proud of the fact that your birth weights are higher. The point is that your death rates are also lower, and the data are very clear on the fact that the two are very intimately related.

There is no question that some of your birth weights may be related to diabetes. On the other hand, the mortality of diabetic babies is higher than that of the general population even though they are bigger babies. So with the reduced mortality which you observed in your studies, you can't argue the statistics both ways.

I am talking about your increased birth weight and your decreased neonatal mortality in that period. If people look at statistics very carefully, they will find out that although there is a 2-3 fold increase in neonatal mortality among people in lower socioeconomic groups which include many blacks, pound for pound there is no difference in infant mortality. Pound for pound, the black baby does just as well as the white baby. Pound for pound, the poor baby from lower socioeconomic group does just as well as the rich baby. The difference is in the low birth weight, and your data point that out so beautifully. Where you have been able to increase birth weight, you have reduced infant mortality, and I think that this is the message from your presentation. This important message, at least to me, should not be diluted by the diabetes problem which is another problem and an important one.

Herbert, J.

In this country, cancers and other chronic diseases that account for most mortality are related to overnutrition, e.g., high fat diets, diets that lead to obesity, etc. On the other hand, there is the problem of hunger which is probably in one sense equatable to protein-energy malnutrition. The complexities of the problems often make it difficult to come up with coherent public policy statements.

Carter, J.P.

I would not equate hunger with protein-energy malnutrition. I think hunger is really a symptom, it is a sensation, but more a symptom of unmet needs. It is wrong to equate the problem of hunger to protein-energy malnutrition and then base policy on that linkage.

Brown, J.L.

I would agree with Dr. Carter and go further to add that we are talking about a chronic sensation reflecting inappropriate diet on a long term basis, and which is generally associated with economic deprivation among the low income population.

Darby, W.J., Jr.

I think this points up a question of semantics and a very important one. The word "hunger" is given different interpretations by various people. Professor John Yudkin had an article a few years ago in which he talked about wants and needs. Many people have a hunger for a cadillac, but they don't necessarily need one. When we talk about hunger, we need to be very precise on what we are talking about and who we are talking to. Malnutrition does not necessarily mean hunger and vice versa.

Williams, W.T.

Dr. Carter, how does one effectively combat the massive amounts of advertising that are detrimental to the growth, development and health of young people? If you pick up any profit-making publication aimed primarily at the black communities, smoking advertisements make up a significant percentage of the "ads".

Carter, J.P.

This is not an easy one. The executive director of the Council on Black Alcoholism and Drug Abuse in Minneapolis, compared the advertisements for cigarettes and alcohol in Ebony and Life Magazines, and found that the former has twice more than the latter. There are some products that are mainly advertised and marketed to blacks. One is malt liquor. My answer to your question is that in the home, parents have to try to provide the kind of knowledge and insulation that will help their youngsters as they go through exposures to all the many advertisements. I am falling back on the assumption that the home would be the best place to insulate a child from the bombardment of false messages. Now this does not mean that they could not benefit from assistance provided by the meager budgets of particular agencies within Health and Human Services, etc. I wish I had a better answer to your question because we are talking about David and Golliath in terms of needed resources, and this is a critical area.

Williams, W.T.

Dr. Jackson, in large inner city populations, particularly in Detroit, Michigan where we have a combination of Blacks, Hispanics, Indians and Appalachian Whites in certain geographic communities, there seems to be much higher rates of diabetes, alcoholism, etc. than you have observed in the reservation.

Jackson, Yvonne M.

It depends on what group you are talking about and also, the degree of aculturation. If you are talking about an Indian population that has been aculturated into the society of that city, then they will probably reflect what is happening there in the city. We have got a very large number of Indians represented in the homeless migrant population, and data on them are probably worse than the data I presented this morning.

The Minority Task Force in Michigan looked at the Indians in Michigan and published a report in 1985 that compares the findings with findings in Indians residing in different reservations.

Darby, W.J.,Jr.

I want to make one very brief comment concerning the question of nutrition education. In attempting to initiate an understanding of the relationship between food and health in the Omaha Indian population, the Swanson Nutrition Center, some years ago, designed some remarkable teaching devices. When they were tried out in the target population, the educational techniques proved to be a failure and had to be revised several times. I use this as an example of how difficult it is to get a nutrition message across without consideration of the sociocultural beliefs of the target group. What was observed in the Omaha Indians applies equally well to other groups, whether they are middle class white Tennesseans, blacks or orientals. A great deal of research and effort is required to develop any kind of effective educational program which will get the message across and also motivate the group to change their behavior. I dare say that those who advertise booze and cigarets are probably more effective in influencing the behavior of people than many of us who preach the virtues of one food over another.

SECOND SCIENTIFIC SESSION

Nutrition Throughout The Life-Cycle

CHAIRPERSON:

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Nutrition in Pregnancy and Lactation

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Introduction

The impact of nutrition on pregnancy and lactation has been examined for decades, although the bulk of research has focused on experimental animals. Observations have been made of pregnant and lactating women in both developed and developing countries but focus on differences among ethnic groups has been limited. Within the United States, data have been compiled on maternal morbidity and mortality, perinatal mortality, % low birth weight (LBW) delivery, and breastfeeding patterns in major ethnic groups residing within the country. Effort has also been made to evaluate potential factors associated with poor pregnancy outcome and lack of interest in breastfeeding among women from various subcultures in North America. Much more limited are studies of pregnant and lactating women in which dietary or supplementation manipulations have been examined to evaluate their impact on the anatomy, physiology, and biochemistry of these reproductive processes. This is especially true when it comes to definition of differences among ethnic groups. Given these circumstances, the present review will focus upon those issues for which data are available. Effort will be made to point to the questions which should be addressed but remain at the moment unanswered in the scientific literature.

Pregnancy: Maternal Mortality and Morbidity in the United States

Recent data obtained from vital records indicate that 6.9 maternal deaths occur annually for every 100,000 live births in the United States (1). This death rate is significantly higher for blacks and others, although the racial difference has decreased over the last 25 years (Figure 1). More accurate data have recently been generated from a confidential pilot surveillance system which was developed jointly by committees in seven states and the Centers for Disease Control; these data are deemed more accurate in that information from death certificates is augmented by clinical descriptions of cases provided by knowledgeable care providers and autopsy records (2). Using this surveillance system, the estimated maternal mortality rate for 1983 was 9.6/100,000 live births with a mortality rate for blacks and others of 16.6/100,000 live births compared with 7.6/100,000 live births for whites. Embolism, peripartum cardiomyopathy, and cerebrovascular accident together accounted for 49% of the maternal deaths. In four (10%) of 39 cases, data were insufficient to arrive at a cause of death more specific than "cardiopulmonary arrest" (Table 1).

The impact of advanced maternal age on pregnancy outcome is of much interest due to the trend toward delaying reproduction in North America (3). Maternal mortality clearly increases with rising maternal age and this phenomenon is especially noteworthy among blacks (Figure 2). Fortunately, maternal mortality rates for women 35 years or older have declined approximately 50% since 1982 (3). Equally fortunate is the observation that among black women, the percent of births to women 35 years of age or older is decreasing rather than increasing as it is among white females (3).

The U.S. Public Health Service's 1990 objective for maternal mortality is a maternal death rate not to exceed five per 100,000 live births for any county or for any ethnic group (4). The rather slow decline in maternal mortality for blacks and others suggests that the 1990 objective may not be met for this group (Figure 1).

The largest data base available on maternal morbidity derives from the Collaborative Perinatal Study of the National Institute of Neurological Disease and Stroke (5). These data were collected between 1959 and 1965 from 12 participating institutions around the United States. The samples were classified into five categories by ethnic group of the gravida: White (n = 25,703), Negro (n = 25,837), Puerto Rican (n = 3,795), Oriental (n = 256), and Other (n = 317). Data from the latter three categories were ultimately excluded from the final report since the numbers in each group were relatively small. Of the 51,540 registrants, 2,106 (4.1%) were lost to the study. The remainder were evaluated prospectively in depth.

The data generated from this massive project are too extensive to review satisfactorily at this time. However, examination of the information on maternal characteristics (Table 2), maternal conditions during pregnancy (Table 3), and complications of pregnancy and labor (Table 4) reveals some striking differences between whites and blacks. The black women were more frequently unmarried, more likely to be 17 years old or younger, less educated, more likely to have many children, and inclined to seek prenatal care later in pregnancy and ultimately complete fewer prenatal visits. No striking differences were noted between groups in maternal conditions during pregnancy but the development of complications during pregnancy or labor varied in several respects. Black women suffered more frequently from pregnancy-induced hypertension and premature rupture of membranes. They were also more likely to deliver LBW infants and as a result, perinatal and neonatal death rates were higher in the black population. Since black women were less inclined to smoke than white women, the greater number of babies delivered by these women cannot be attributed to this factor. While diet and nutrition were not evaluated in this study, it is possible that nutritional inadequacy may have contributed to the higher rate of low birth weight among blacks. This conclusion is justified based on the demographic data which imply a lower socioeconomic status in this group.

Perinatal Mortality and Low Birth Weight Delivery

Since the publication of the Perinatal Collaborative Study in 1972, effort has continued to monitor pregnancy outcome of women from virtually all subsets of the U.S. population. With regard to low birth weight among various racial/ethnic groups (6), the incidence remains substantially higher among blacks than among any of the other subgroups evaluated (Table 5). Even though improvement has been apparent since 1970 (Table 6), 12.4% of black babies born in 1984 weighed less than 2500 grams (6). Japanese babies have about the same rate of low birth weight as white infants but Chinese babies have an even lower proportion, 5.1% in 1984. Other races were slightly higher but none higher than 7.3% (7).

The higher percentage of low birth weight black infants compared with other groups has remained constant for the past 20 years (8,9). However, the reasons for this increased risk are not completely understood. Maternal age may account for part of it. Teenage mothers are at high risk for producing LBW infants, and black mothers are more likely to be teenagers than are mothers of other ethnic groups (10). However, according to Geronimus (11), neonatal mortality is higher for blacks at every maternal age and in fact, black primiparae above 23 years of age experience higher neonatal mortality than most black and white teenagers. The hypothesis that excessive LBW or neonatal mortality is due to greater frequency of teenage childbearing among blacks is refuted.

Low level of education, often a marker of socioeconomic status, is another risk factor for LBW. However, although black mothers are generally less educated than white mothers (Table 7), when matched for both age and education, blacks are still at higher risk of delivering LBW infants (12). Similarly, black women are more likely than white women to delay initiation of prenatal care (Figure 3) (13), but when prenatal care is held constant, black women are still at increased risk of LBW outcome. When maternal smoking, stature, pregnancy weight gain, all combinations of age and parity, and history of previous LBW infant are controlled, black women still have an increased risk of LBW delivery.

In contrast, Mexican-Americans of similar economic and sociodemographic characteristics as blacks have a very low incidence of LBW infants (Table 5) (12), which may reflect problems of data reporting or cultural differences (e.g. lower incidence of childbearing among unmarried women, different dietary practices and family support structures). Although the cumulative effects of poverty and social neglect and the interaction of these effects with biologic factors have understandably played a significant role in the resultant black-white LBW differences, other important factors remain to be defined.

On a worldwide basis, of the 127 million infants born in 1982, 16% -- some 20 million -- had low birth weight (14). Variations between and within geographic regions remain considerable. The incidence of low birth weight, by region, ranges from 31.1% in Middle South Asia and 19.7% for Asia as a whole to 14.0% in Africa, 10.1% in Latin America, 6.8% in North America and 6.5% in Europe (Table 8). In countries where the proportion of low birth weight infants is low, most are preterm. In countries where the proportion is high, the majority of low birth weight infants suffer from fetal growth retardation. The causes of fetal growth retardation are multiple and interrelated and include low maternal food intake, hard physical work during pregnancy, and illness, especially infections. Short maternal stature, very young age, high parity, and close birth spacing are all associated factors. While suboptimal nutritional support may be associated with all of these factors, it is impossible to quantitate the significance of this variable in the etiology of low birth weight in any setting.

Factors Associated with Adverse Pregnancy Outcome

Many factors have been found to be associated with adverse pregnancy outcome (Table 9). Some can be identified before pregnancy occurs, and possible interventions can be implemented before the prenatal period. In addition to the distinct medical risks which predate pregnancy or develop during the gestational period, a number of risks may be classified as behavioral, environmental and demographic. These require nonmedical interventions like nutrition counseling or nutrient supplementation.

The significance of maternal nutrition in the reproductive process is now well recognized and unlike some genetic and environmental variables, women (and their partners) have control in determining their nutritional status. Not only does maternal diet during pregnancy impact upon fetal development but diet before pregnancy may affect fertility and determine the adequacy of nutritional support for the embryo (15).

Achieving a nutritional status supportive of fertility does not necessarily mean that nutritional status is optimal for support of a pregnancy. In fact, many women today conceive while consuming a nutritionally unbalanced diet. Unfortunately, pregnancy is often confirmed well after the critical period of embryonic development has passed. If, in fact, maternal nutritional deficiency or excess is associated with the production of congenital malformations, then attention to diet after pregnancy is established is clearly too late.

With regard to low pre-pregnancy weight of the mother, underweight status has been shown to be associated with higher risk of adverse outcome. For example, Edwards and colleagues (16) reported that underweight women had significantly higher rates of cardiac/respiratory problems, anemia, premature rupture of membranes, and endometritis. Prematurity, low birth weight, and low Apgar scores were significantly more frequent in the infants of underweight women. The mean birth weight of the infants of these women was 231 grams less than that of infants of control subjects. Observations of the infants during the first year of life showed that those who were of appropriate weight for gestational age were more likely to be below the 25th percentile for weight correlated with length at 12 months of age if their mothers were underweight prior to pregnancy. Limited data also suggested delayed neurologic development in these babies.

The incidence of low prepregnancy weight has recently been evaluated in a group of pregnant women in Texas (Personal communication, University of Texas Health Sciences Center at Dallas, 1986). The women were primarily black (45.7%) and Latin American (43.4%), and approximately equal proportions of the women were single (44.6%) and married (45.7%). Formal education ranged from 0 to 15 years with over half of the women having less than 12 years of schooling. Assessment of maternal weight for height revealed that a significant number of women had weights less than 90% of standard in both the nonpregnant state (25.2%) and at the time of their first prenatal visit (14.8%). How representative these data are of blacks and Latin Americans in other settings is unknown. It can be concluded, however, that low prepregnancy weight status is a significant problem rectified only by proper nutritional support.

Although the relationship between mother's weight gain during pregnancy and the outcome of pregnancy has been explored in numerous studies, only one has been national in scope. This recently published investigation examined the association between maternal weight gain during pregnancy and pregnancy outcome. Data were obtained from the 1980 National Natality Survey (NNS) and the 1980 National Fetal Mortality Survey (NFMS) conducted by the National Center for Health Statistics (17). The NNS is based on a probability sample of all live births occurring in the United States in 1980. The NFMS is based on a probability sample of fetal deaths with a physician's estimate of gestation of 28 weeks or more or delivery weight of 1,000 grams or more if the physician's estimate was not reported. The total sample of 3581 women included 2917 whites and 554 blacks.

Although current obstetrical guidelines state that usual weight gain in a normal pregnancy should be 22-27 pounds, the recent NNS/NFMS report indicated that one out of five white mothers and one out of four black mothers whose pregnancy extended to at least 40 weeks gained less than 21 pounds. Overall, 23% of women giving birth in 1980 gained less than 21 pounds during their pregnancy and 12% gained less than 16 pounds. These data provoke concern since maternal weight gain was inversely related to percent LBW delivery and fetal mortality up to 26-35 pound, the range associated with optimum outcome (Figure 4).

There were other noteworthy observations reported in the NNS/NFMS 1980 publication (17). Black mothers were 50% more likely than white mothers to gain less than 21 pounds (33% compared with 22%) and they were twice as likely to gain less than 16 pounds (20% compared with 11%). The average weight gain of white mothers was 29.1 pounds, 2.3 pounds higher than that of the black mothers. Large racial differences were found to exist even after taking into consideration the shorter gestational period of black births and variation in prepregnancy weight. As one might expect, percent low birth weight was more than twice as high among blacks (12.5%) than among whites (5.6%). The same was true for fetal deaths with rates of 7.2 and 4.8 (per 1000 live births) for blacks and whites, respectively.

Other relevant data derive from the Pregnancy Nutrition Surveillance System which processed over 26, 000 records from 14 states and the District of Columbia during 1983 (18). These data come from maternal and child health, WIC,and other health programs providing prenatal care. Low hematocrit levels were evident in 25.8% of the women while 17.3% had low hemoglobin levels. Black women had by far the highest prevalence of anemia with 35.9% having low hematocrit levels and 26.6% having low hemoglobin levels. Since anemia is associated with increased risk of poor pregnancy outcome, all pregnant women, but especially blacks, should be closely monitored so that early detection of this abnormal hematologic state can be accompanied by appropriate dietary and/or supplement intervention.

The Value of Individualized Nutrition Counseling

That nutrition intervention may help improve maternal health and pregnancy outcome is suggested by a recent study of Orstead et al (19). These workers compared the value of a single prenatal nutrition class for pregnant women with individualized nutrition counseling during the latter half of pregnancy. The patients under study were Black (70%), Asian (10%), Hispanic (5%), and Caucasian (15%). The women receiving the nutrition counseling gained about 6 pounds more weight and had fewer low birth weight infants (4% vs. 13%) than the controls. When the cost of the care of the extra low birth weight infants in the control group was compared with the cost of the dietitians' time in individualized counseling, a benefit-to-cost ratio of 5:1 was found. It would therefore appear that good nutrition counseling may not only improve maternal and infant well-being but is also cost-effective in our current system of health care delivery.

Nutrition Supplementation Programs

The value of providing nutritional supplements to pregnant women is an issue subject to much debate. A number of supplementation studies have been conducted during the past three decades in both developed and developing countries. In general, one can summarize the results as follows: The worse the nutritional condition of the mother entering pregnancy, the more valuable the nutritional supplement will be in improving pregnancy course and outcomee (15). Modest improvement in birth weight and perinatal mortality has generally been seen when malnourished pregnant women in developing countries are provided a nutritional supplement containing extra calories, protein and micronutrients. Supplementation trials in developed countries have generally shown limited if any improvement in measures of pregnancy outcome. Since many disadvantaged women in developed countries are relatively well nourished, it is not surprising that providing extra food yields little measurable change in maternal or fetal well-being.

An example of a major supplementation trial in the United States is the provocative New York Study (20,21). The investigators were concerned with the high rate of low birth weight infants born to poor black women in Harlem who were presumed to be poorly nourished. Attributes of low income black women who were at especially high risk of bearing LBW infants were first identified in preliminary studies. A target population was selected on the basis of these attributes.

Briefly, this randomized, partially double-blind, controlled trial of prenatal supplementation carried out among poor women in a typical inner city in the United States yielded paradoxical results. The data suggested that the women used the beverage appropriately. The beverage had no significant effect on birth weight. Women receiving a balanced protein-calorie product had an advantage in birth weight of 41 grams over controls and fewer low birth weight infants. Those women ingesting a high protein supplement had a disadvantage in birth weight of 42 grams. They also had high rates of very premature delivery and neonatal death and, among prematures, retarded growth. Conditional effects were present in both directions. Women with previously low birth weight infants responded adversely to the high protein supplement and women who were heavy smokers responded favorably to both supplement products. Among the majority who gave birth at term, the birth weights of the supplemented groups were in the expected direction, but the difference between the extreme groups was only 24 grams, well below the difference anticipated in the study. Conclusions drawn from the New York Study were few but it appears that in United States' populations that are at high risk of low birth weight, nutritional supplementation has modest effects on birth weight except in heavy smokers.

These data generated from the New York study should not be used to justify discontinuation of the WIC program in the United States. Virtually all studies conducted to assess the effectiveness of the WIC program have demonstrated positive results. For example, Schramm (22) reported observations in Missouri of women of comparable socioeconomic status who were or were not participating in the WIC program. Among the 3251 whites, percent LBW was 9.2 for WIC participants and 10.1 for non-WIC women; among 4377 blacks, percent LBW was 11.9 for WIC participants and 14.7 for non-participants. Metcoff et al. (23) reported in a similar comparison of WIC and non-WIC participants in Texas that the WIC effect on birth weight for the non-black babies was +118 grams but for the black infants it was 199 grams. The large national evaluation of the WIC program conducted by Rush and reported in 1986 (24) again demonstrated that participation in WIC was a positive experience, especially for those poor women with limited educational background.

Rush examined this latter group of women (low income with less than 12 years of education) and reported a reduction in low birth weight of 20% among WIC participants. Using this figure, impact of the WIC program was estimated by considering the 110,601 women with less than 12 years of education who were on public assistance in 1982. The LBW rate of 115/1000 live births in 1982 would have been reduced to 91/1000 live births by WIC participation. This reduction of 23 low birth weight infants for every 1000 live births was calculated to have an important impact. This includes: 1) an estimated decrease of 10% in infant deaths, and this translates to a decline of 2.3 infant deaths per 1000 live births or 254 infants in this high-risk group of women alone. 2) an estimated savings in direct medical costs for care of the 254 LBW infants delivered by this high risk group of $38,122,814 in 1983 dollars. These figures are generated by adding together the following costs (25):

1. intensive care hospital costs for mothers and babies ($13,616/LBW baby)	$ 34,639,104
2. rehospitalization of 20% of surviving LBW infants ($5,580/baby)	2,840,220
3. long-term care of 18% of the surviving LBW infants ($1,405/baby)	643,490
Total	$ 38,122,814

Thus, for this one cohort alone, a significant decrease in neonatal morbidity and mortality was accompanied by a marked reduction in health care costs. This positive effect of WIC may be exclusively through nutritional supplementation or, more likely, through a combination of nutritional supplementation and the other two components of the WIC program for pregnant women, prenatal care and nutrition education.

The Practicalities of Nutrition Consultation for Women of Different Subcultures

Effective nutrition counseling is clearly associated with in-depth understanding of existing dietary practices. While these practices may be highly individual, patterns are seen within most ethnic/racial subgroups which influence choices women make about specific foods and food combinations (26). Even though the United States has become a melting pot as far as ethnic distinctions are concerned, recognition of several basic themes is relevant.

The Typical Middle-class American Diet

This is the diet that the majority of Americans eat and its characteristics are worth noting to provide perspective on other diets used by Americans. Having its origin in Great Britain, it has added dishes and techniques of preparation from many other countries during the last 200 years. Today, many of these originally non-British dishes have been so fully integrated as to be, for middle-class Americans, often labeled "ethnic". In its nutritional features, this diet is but one variation of the affluent diet of industrialized countries (27).

The foods and food combinations viewed as particularly American share certain design themes, including the heavy use of sugar, the frequent combination of salt, and sweet or sour (e.g. ham and pineapple, turkey and cranberry sauce, salted nuts on ice cream), the heavy use of raw foods to make salads (again in odd combinations) and an emphasis on the visual presentation of "perfect" fruits and vegetables and in abundance. Of major importance is the centrality of meat in the diet. To many Americans, meat is the meal and everything else is peripheral.

The orthodox middle-class American diet has recently been labeled unhealthy at least in the long run by scientific experts. The Dietary Guidelines for Americans recommend that Americans increase their consumption of unrefined carbohydrates and radically reduce their use of fat, sugar and salt to combat the chronic and degenerative disorders that have been linked with consumption of the affluent diet (28).

Black Americans

It is somewhat artificial to separate black from white Americans in describing diet, for black Americans typically consume a regional cookery that does not differ much from that of their white neighbors (26). Several factors, however, are worth noting. First, there are more low-income black people, and low incomes are often associated with less nutritious diets. Second, many black people are lactose intolerant and consumption of dairy products is therefore lower in this group. Finally, several design themes unfamiliar to many biomedical practitioners are widely used by black Americans in selecting their diets. These may have important nutritional implications. Probably the best example here is the belief that the quality and character of the blood ("hot" and "cold") are directly related to diet. A healthy body is believed to display a balance of hot and cold (humors). "High blood" and "low blood" are thought to be corrected through dietary manipulation. For example, blood is considered hot. "High blood" (high blood pressure) can cause stroke and is believed to come from eating too much rich food or red meat, such as pork. The treatment is believed to be dietary, including use of astringent and acidic foods such as vinegar, lemons, pickles, or epsom salts to "open the pores and let the excess be sweated out." These ideas are relevant to the prenatal diet because the pregnant woman is "hot". She must control her diet to protect herself from "high blood", and this may involve avoiding meats and high-caloric foods or increasing the intake of sodium-rich foods. Additionally, "high blood" is terminologically confused with high blood pressure, so that individuals holding this misconception who are told their blood pressure is high may ingest items with a high sodium content as a form of self-treatment or eliminate meat from a diet which may already be protein-deficient (29).

Mexican Americans and Puerto Ricans

While the diets of Mexican Americans and Puerto Ricans differ, the central theme for food selection--the hot-cold theory of health--is shared by many in both subcultures. Although by no means used by all, it is important for many. In this system, foods have inherent (symbolic) temperatures, such as cold, neutral or hot. A meal provides a balance, and a person can get sick by eating foods whose temperatures are wrong for him or her. A pregnant woman is "hot" - hence, she must avoid both very hot and very cold foods. In practice this means that some pregnant Mexican American or Puerto Rican women will avoid chili peppers and some salty, fatty or sweet foods ("hot") and acidic, sour or fresh foods ("cold"), such as lemons, tomatoes or watermelon (30). To adherents of the hot-cold theory of health, foods are medicines. By dietary management one maintains good health and can treat illness. The astute health practitioner can integrate biomedical beliefs with hot-cold beliefs to improve compliance.

Asian Americans

Oriental food traditions are ancient and complex and show much conservatism (32). Diet is intimately associated with health. Oriental cookery is characterized by mixed dishes with the components cut small. Preparation is relatively lengthy but cooking time is often brief. Characteristic foods include rice, wheat, pork, chicken, a variety of vegetables (served barely cooked), eggs, various soy preparations, and tea. A favorite sauce combines sweet and sour flavors. Salt intake is high and fat is low. Milk use is rare and adult lactase deficiency is widespread. The Chinese largely maintain the same food patterns when in the United States (33). This is also true for the Vietnamese who maintain a strong preference for Vietnamese foods even after years of residence in the United States (34). Counseling strategies with pregnant women should address culturally based food preferences whenever possible.

Unusual Prenatal Dietary Practices

A number of researchers have attempted to describe the idiosyncrasies of dietary practices of pregnant women from various ethnic/racial groups. Possibly, the single most common belief is that the mother's diet can mark her child prenatally. Typically, marking is associated with food cravings: 1) Overuse of a craved food may mark the child. Frankel (35) noted the case of a black woman who attributed her daughter's peach allergy to her own excessive use of peaches while pregnant. 2) More often, unsatisfied cravings are thought to cause trouble. Pregnant black American women (36) and Greek women (37), for example, are permitted and indeed expected to go knocking on neighbors' doors seeking the single food that will satisfy a craving. A birthmark on a baby is believed to take the shape of the unavailable craved food: strawberry- or cherry shaped marks (36, 38, 39), chicken-drumstick marks (35) and so forth. 3) Some substances are thought to prevent marking. This explanation was given for pica among black Americans interviewed by Edwards et al. (40). Since marking beliefs may help the pregnant woman get food to satisfy her cravings, and since cravings may reflect real physiological needs, marking beliefs may be health engendering. There is no evidence that they are deleterious.

Many foods are avoided during pregnancy in the hopes of ensuring an easier delivery. Many peoples avoid protein foods (41) or salty or sweet foods (35, 42), while others simply try not to gain too much weight (39, 43). Needless to say, aggressive effort toward this latter end may impact adversely on pregnancy outcome. Most other conscious food avoidances have minimal impact on overall nutritional status of the mother.

Pica is an interesting phenomenon characterized by the compulsion for persistent ingestion of unsuitable substances having little or no nutritional value. Pica of pregnancy most often involves consumption of dirt and/or clay (geophagia) or starch (amylophagia). However, compulsive ingestion of a variety of non-food substances has been noted, such as ice, burnt matches, hair, stone or gravel, charcoal soot, cigarette ashes, mothballs, antacid tablets, milk of magnesia, baking soda, coffee grounds, and inner tubes of tires. The practice of pica is not new nor is it limited to any one geographical area, race, creed, culture, sex, or status within a culture.

Surveys in the southern United States provided some of the first data to identify current pica practices. A study in 1950 indicated that 41% of 331 pregnant black women in rural Mississippi ate starch and 27% ate clay (44). Of 30 whites interviewed, 10% and 7% reported starch and clay eating, respectively. A 1959 survey of 86 patients in an Alabama hospital showed that clay eating was reported by 49%, starch by 27%, and both substances by 7% (45). In other studies as many as 50% of women attending health department clinics in the South consumed starch and 25% consumed clay.

Several researchers have documented the continued practice of pica in the 1970's and 1980's, although at lower levels than previously observed (46). A recent evaluation of pregnant black women in Washington, D.C. indicated that 3.5% acknowledged eating non-food items while pregnant, 9.2% ate non-food items such as cornstarch, clay or ice before becoming pregnant and 38.7% stated that they saw their mothers or other pregnant women eat such non-food items; 34.5% indicated they ate such items when they were growing up (47). Although pica appears to be most common among pregnant black women, it is reported to occur among other ethnic groups in the United States and elsewhere.

The medical implications of pica are not well studied although several speculations have been put forward. The displacement effect of pica substances could result in reduced intake of nutritious foods, leading to inadequate dietary intakes of essential nutrients. Alternatively, substances that provide calories (e.g. starch) could lead to obesity if ingested in amounts above the usual dietary intakes. Some pica substances may contain toxic compounds or quantities of nutrients not tolerated in disease states. Some pica substances interfere with the absorption of certain mineral elements (such as iron). Other less commonly reported complications of pica follow (15):

1. Congenital lead poisoning secondary to maternal pica for wall plaster
2. Tender, irritable uterus with dystocia associated with fecal impaction from clay ingestion
3. Fetal hemolytic anemia caused by maternal ingestion of mothballs and toilet air fresheners
4. Parotid enlargement and gastric and small bowel obstruction from ingestion of excessive laundry starch
5. Parasitic infection from ingestion of contaminated soil or clay

The etiology of pica is poorly understood, although several proposals have been put forth. One theory suggests that the ingestion of nontraditional substances relieves nausea and vomiting. The example of the dog who will eat dirt or grass during illness is often used to illustrate this theory. Recent research indicates that pica is a normal behavioral response to gastrointestinal tract upset in rats. It has also been hypothesized that a deficiency of an essential nutrient such as calcium or iron results in the eating of non-food substances that contain these nutrients. When prenatal patients were questioned concerning the practice of pica, a variety of answers were given:

1. A taste for clay
2. Clay kept the baby from being marked at birth
3. Nervous tension was relieved
4. Starch made the newborn lighter in color
5. Starch helped the baby to "slide out" more easily during delivery
6. Clay quieted hunger pains
7. Clay and starch were pleasant to chew
8. Social approval of pica

Many of these reasons are based on superstition, custom and tradition, or practices that are passed from mother to daughter.

In summary, nutritional counseling of pregnant women should include attention to the possibility that unusual dietary practices or consumption of non-food items may be taking place. In so far as such practices have no potential adverse impact on the course and/or outcome of the pregnancy, they may be noted but not necessarily discouraged. When practices are observed that present the potential of harm to either mother or fetus, appropriate recommendations should be provided for alternative food choices or cessation in intake of harmful non-food substances.

LACTATION

Lactation is obviously the normal physiological process designed for nourishing the infant. The process is a nutritionally demanding one for women although the specific requirements are as yet uncertain. Available data suggest that nutrient needs of the lactating mother relate directly to the amount of milk produced (15). Differences in requirements among various ethnic/racial groups have not been reported although chronically undernourished women in developing countries appear to successfully nurse their infants at the expense of their own nutrient reserves. In so far as the health of the infant is concerned, breastfeeding is believed to provide substantive advantages. Among the 1990 Health Objectives of the Nation is the following: "The proportion of women who breastfeed their infants at hospital discharge should be increased to 75%, and the percentage of those still breastfeeding at 6 months of age should be increased to 35%." (4)

Trends in breastfeeding in the United States

Breastfeeding was the dominant mode of infant feeding in the United States until the early 1940's (48, 49). It dropped steadily in popularity until about 1972 when the trend against breastfeeding began slowly, but steadily, to reverse. Specifically, in the early 1940s, about 75% of all babies were breastfed. This is understandable since commercial infant formula was not widely marketed in the United States until after World War II. Ten years later--in the early 1950s--fewer than half of all babies were breastfed. This figure dropped to fewer than one third of all infants by the early 1960s to a low of about 22% to 25% in the early 1970s (50).

The first evidence of the reversal of the downward trend in breastfeeding appeared in the Ross Laboratories 1972 survey (51), which showed an increase of 3 percentage points to 28% over the low of 25% in 1971. By the mid-1970s, at least one third of all infants were breastfed and this increase in the incidence of infants ever breastfed continued steadily into the 1980s.

In the mid-1950s and probably somewhat later, black infants were more likely to be breastfed than white infants. However, the decline in breastfeeding during the 1960s was more rapid in black women than in white women, so that by 1970 white babies were more likely than black babies to be breastfed. In one study of first births, 59% of black babies, as compared with 49% of white babies, were breastfed in 1951 to 1955. By 1966 to 1970, only 14% of black babies were breastfed as compared with 29% of white babies (52). This accelerated decline in breastfeeding rates among black women in the 1960s has carried over into the recent post-1970 shift favoring breastfeeding. Although there is evidence that the incidence of breastfeeding increased in both black and white mothers in the 1970s, this trend was stronger among white women. According to one estimate in 1975, 38% of white infants, as opposed to 17% of black infants, were "ever breastfed" (53).

There are no national estimates of infant feeding trends among Hispanic women in the 1970s. However, data are available for the 1950s and 1960s. In the 1950s, the incidence of women breastfeeding their infants was comparable to that of the black community during the same period--about 58%. This rate declined to about 35% by 1966 to 1970, slightly higher than the rate for white women (29%) during this period (52).

Most of what is known about infant feeding patterns in recent years comes from the Ross Laboratories National Mothers' Surveys. Although the Ross surveys suffer from several significant methodologic limitations, they provide the best available estimates of current national experience. In fact, the breastfeeding rates reported by Ross for 1980 (54%) are similar to those identified in the National Natality Survey (58%) (54) and the National Survey of Family Growth (52.5%) for the same year (55).

The major limitation of the Ross Laboratories' data is that, even with statistical adjustments for the disproportionate numbers of poorly educated and disadvantaged women who did not respond to the questionnaire, the experience of disadvantaged women is probably underrepresented in the reported rates. Moreover, before 1983 the effects of race and ethnicity were not investigated in the Ross surveys, nor was attention given to whether the birth was legitimate or illegitimate or to whether the mother was married or not.

Even still, the Ross Laboratories' data do describe national trends. Specifically, these data show a continued increase in the incidence of breastfeeding from 1976 to 1982 and an apparent plateauing of the trend between 1982 and 1984 (Figure 5). After adjustments for underreporting by subsets of the population, approximately 62% of babies left the hospital breastfeeding in 1982. This percentage is virtually unchanged in 1983 (61.4%) and 1984 (62.5%). Durations of breastfeeding rates were similarly unchanged between 1982 and 1984 (37.5% to 37.0%). When the 1983 and 1984 data are reweighted to adjust for nonresponse by race and ethnicity (variables unique to the 1983 and 1984 surveys), the rates referred to above are revised slightly downward. Specifically, breastfeeding of infants in the hospital slips to 58.4% from 61.4% in 1983 and to 59.7% from 62.4% in 1984. Similarly , breastfeeding of infants at 3 and 4 months slips to 34.1% in 1983 and 34.8% in 1984 (51).

Reweighted 1983 and 1984 data provide the best approximations of recent rates for the nation by demographic features (Table 10). Differentials in the incidence of breastfeeding by race, maternal education, maternal age, and geographic region continue to be great. In 1984, breastfeeding was most prevalent in the Western states among college educated women and among white mothers. The incidence of breastfeeding was lowest among black women, women under 20 years of age, and the less well educated.

While the Ross data make clear the relatively low incidence of breastfeeding among disadvantaged women and minorities, it is likely that the Ross rates, if anything, overestimated the actual breastfeeding experience among disadvantaged subgroups. Smaller studies of local populations support this conclusion. For example, Rassin et al. (56) examined a Texas population characterized as consisting generally of young, unmarried, low-income, and racially diverse mothers (Anglo-Americans, Black Americans, Mexican-Americans). Within 48 hours of birth, 358 women completed questionnaires. A telephone follow-up was conducted with 64% of the population at 6 months postpartum. Twenty-seven percent of the mothers were breastfeeding during the hospital stay. At the 6 month follow-up, 79% of those breastfeeding women had stopped breastfeeding. There were significant differences in the proportion of women initially breastfeeding within various demographic categories (Table 11). The most important demographic factor identified in this study was ethnicity. American black women breastfed much less frequently than American white women (9.2% versus 44%) and among the Mexican-American women, 23% breastfed. The rate for blacks was also much lower than that reported for blacks in the 1983 Ross survey. The importance of ethnicity is also supported by the findings of a recent study by Smith et al. (57), in which Hispanic women of Mexican origin, with more than 12 years of education, breastfed their infants at a rate of 33% compared with a rate of 53% among Anglo-Americans with comparable education.

Finally, several research groups have attempted to evaluate the breastfeeding patterns of the disadvantaged women who participate in WIC. Martinez (Personal communication) used the 1984 Ross survey data and weighted it for race and ethnicity (Table 12). Overall, 39% of WIC participants in the 1984 survey were breastfeeding their infants in the hospital and 13% of the infants were still receiving breast milk at 5 to 6 months. Breastfeeding rates in the hospital ranged from a high of 69% in the Pacific states to a low of 25% for black WIC participants.

At least two other published accounts of infant feeding practices among limited samples of WIC participants reported breastfeeding rates that are comparable to the Martinez-Ross findings (58, 59). While substantial differences are seen from one geographic region to another, no data are available to support a conclusion that differences exist among ethnic/racial groups in the biological capacity to lactate.

Factors Affecting the Decision to Breastfeed

If biological factors cannot explain distinct differences in breastfeeding incidence among subsets of the U.S. population, then psychosocial and related influences may be important. Factors affecting breastfeeding behaviors have been examined in a number of populations in both developed and developing societies. Data are available on knowledge, attitudes, social support, and other influences on the "breast versus bottle" decision-making. Such data are helpful in identifying target groups for programs designed to increase breastfeeding rates and they also contribute to the design of effective individual and group interventions.

Early studies of various sociodemographic groups suggest that women who breastfeed their infants are more knowledgeable about the benefits of breastfeeding than women who feed their infants formula (60-61). Good sexual adjustment has been positively correlated with breastfeeding (62) while embarrassment and other emotional barriers have been more commonly noted among women who bottle-feed their infants (63, 64). Positive attitudes about breastfeeding and beliefs about its convenience and its benefits for the baby have also been reported as influencing factors (61, 62).

Social influence is substantial and network factors such as opinions of husbands, relatives and friends also appear to influence women's decisions about breast- and bottle-feeding. Early studies suggest that positive attitudes and support from these sources is associated with greater incidence of breastfeeding (64, 65). Relatives and friends who successfully breastfeed their infants also contribute to a woman's decision to breastfeed.

In a recent study by Sullivan and Jones (67), the breastfeeding decisions of 181 black women (median age 21 years) from a large public teaching hospital were examined. An early prenatal decision to breastfeed (EDB) was made by 42 women, and an early decision not to breastfeed (EDNB) was made by 115 women. Many more women in the EDB group than in the EDNB group had been breastfed as children and their own mothers or other relatives were considered an influence in their decision to breastfeed. More women in the EDNB group said that a man or a friend helped them make their decision and more remembered a health care professional discussing breastfeeding with them prenatally. Most women in the EDB group perceived breastfeeding as good for their baby, whereas more women in the EDNB group perceived breastfeeding as inconvenient, uncomfortable or embarrassing for themselves.

The women in the Sullivan/Jones study (67) were classified after giving birth into adaptors (42 women) or rejectors (139 women). More adaptors than rejectors reported having been breastfed as children and remembered participating in prenatal preparation. Of the women who discussed breastfeeding with a health professional, more rejectors talked with a physician and more adaptors talked with a nurse or dietitian. Adaptors had more social support during birth and more infant contact than rejectors. Rejectors' infants had more complications than adopters' infants.

After 6 weeks, adaptors were reclassified into continuers (breastfed for at least 6 weeks) and discontinuers (breastfed less than 6 weeks). Continuers had more social resources, were more likely to have been breastfed as children and were less likely to plan to work or attend school after giving birth. The authors suggest that programs to increase awareness of breastfeeding should be targeted at grandmothers as well as at pre-childbearing and pregnant women, because of the influence grandmothers have on breastfeeding decisions.

Postpartum experiences also influence the infant feeding practices ultimately adopted by families from all ethnic groups. Postpartum factors affecting breastfeeding have recently been examined among women of Mexican origin or descent in Los Angeles (68). Data on breastfeeding intentions and behavior were collected in prenatal and postpartum interviews with 518 women delivering in two Los Angeles hospitals. This sample consisted of young women, most of whom were born in Mexico and relatively recently arrived in the United States. Eighty-two percent of the women interviewed prenatally planned to breastfeed. These prenatal intentions were maintained in one hospital but dropped sharply in the other. The greater the number of hours a day with the baby in the hospital, and earlier initiation of breastfeeding were associated with the hospital where prenatal breastfeeding intentions were more likely to be carried out.

In another study of low income WIC mothers (black, white, Puerto Rican), the 26% of women who chose to breastfeed were generally older and had completed more years of education (69). Jamaican women were more likely to breastfeed (73%) compared with white women (33%), Puerto Rican women (11%), and black women (20%). Women who breastfed their child were more likely to have been breastfed themselves. Having at least one friend who breastfed her infant and having support of the baby's father were also related to breastfeeding. By contrast, the majority of women who fed their infants formula had not discussed their feeding choices with the baby's father. Sixty-seven percent of women who breastfed their infants had received advice from health professionals that breastfeeding was better, compared with only 13% of the women who fed their infants formula. The most common reasons given for not choosing to breast`feed were that embarrassment was substantial (29%) and that they were forced to return to work (9%). The most common reasons given for choosing to breastfeed were the nutritional advantages, "its best for the baby" (27%), discussion with family members or friends (19%) and family or cultural tradition (16%).

Two additional studies have explored beliefs and social networks across different ethnic groups. In 1976, Bryant (70) interviewed economically disadvantaged Cuban women (n = 28), Puerto Rican women (n = 28), and Anglo-American women (n = 20) in Florida to explore social network influences on infant feeding practices. Overall, 36% of the women had breastfed one or more of their children. For youngest children, 35% of Anglo-American women were breastfeeding compared with 10% of the Puerto Rican women and 12% of the Cuban women. Friends, relatives, and neighbors were considered as influential as health care professionals as sources of information and advice, for the Anglo-American respondents. Puerto Rican and Cuban women more frequently considered the maternal grandmother as most important of the network members. The husband was more often a participant in infant feeding decisions for Anglo-Americans but not for the other groups.

Another recent study also found ethnic differences in sources of social influence and support. Rassin et al. (56) examined a group of young, mostly unmarried, low socioeconomic status women and found social support and social influence were very different in their relationship to breastfeeding and varied with marital status and ethnicity. It was found that the male partner is the single-best predictive social support variable and previous breastfeeding is the best social influence variable for Anglo-American women. Of interest, the grandmother is a significant negative influence on breastfeeding among married Anglo-American women. By contrast, for Mexican-American women, the grandmother is the best predictive social support variable. Social influence variables of grandmother, friend, and doctor combine to form the best predictive model for Mexican-American women. For black women, "best friend" is the explanatory variable of choice.

One final source of information about low-income women's decision-making comes from a study conducted as part of the national market research for the "Healthy Mothers, Healthy Babies" campaign (71). Focus group sessions were held with 130 low income black and Mexican-American women from rural and urban areas. These authors reported that the Mexican -American viewed breastfeeding as backward and not Americanized. For both groups of women, concerns about breastfeeding included its effect on breast shape and the beliefs that it was embarrassing to nurse in public, that returning to work made breastfeeding unrealistic, that one could not alternately breastfeed and bottlefeed, and that if the baby did not nurse during the day the milk would "go bad".

For health information in general, these women believed that the public health clinic was their primary source. WIC program workers were highly respected sources of information. Although the physician was deemed the most credible source of health information, nurses were their key contacts and were seen as good information sources, especially for personal matters. Family and friends were noted as dominant influences, and male partners were sources of encouragement and support. Pamphlets and television were the nonpersonal influential sources of health information.

Overall, Adair (72) suggests that this pattern of influence is specific to lower-income women. In her ethnographic study, she noted that women of higher socioeconomic status cited more varied sources of information about feeding, including prenatal classes, and that their reading material tended to be specific books or articles on breastfeeding. Clinic patients used personal networks of friends and relatives and their reading material was most often pamphlets.

Interventions to Promote and Support Breastfeeding among Blacks and Others Minorities

While research on breastfeeding attitudes is inconsistent, particularly in its attention to variations between ethnic subgroups, a great deal is known about attitudes toward breastfeeding, barriers, and the reasons for not initiating breastfeeding or for early discontinuance. Many of these reasons, such as the need to work and hospital practices, present problems for women from all ethnic groups, although cultural values and institutions will influence the way these barriers are managed. Attitudinal research and research on breastfeeding incidence remain important but are of greater value when combined with research aimed at reducing hospital barriers to breastfeeding and developing and testing high quality intervention programs.

Cultural norms guide decisions about breastfeeding and influence support for breastfeeding. Cultural attitudes must be taken into account in the design of intervention programs. Despite the importance of ethnicity, education, and socioeconomic status, other factors need serious attention. These include postpartum participation in the work force, general U.S. attitudes about breastfeeding in public, hospital practices, and health care providers' knowledge, attitudes and behaviors. These areas must be addressed in order to facilitate breastfeeding for women in all ethnic groups.

Several intervention studies involving disadvantaged populations have been recently reported. The least successful of these is a study of WIC program participants which focused only on prenatal education and its effects on the feeding method at time of discharge and at two months postpartum (73). The educational intervention was a series of five pamphlets on breastfeeding specifically designed and tested for use with this audience. Forty-five women were randomly assigned to receive all five pamphlets or to receive no intervention. Knowledge and attitudes about breastfeeding were assessed by interviews before and after the intervention. The pamphlets significantly increased knowledge scores among women who intended to bottle-feed and those who were undecided but had no effect on attitudes, initiation or duration of breastfeeding. Overall, 45% of the women sampled were breastfeeding their infants at hospital discharge.

Much more successful was an in-hospital intervention program for an inner city poor population in Texas (74). Sixty breastfeeding mothers were randomly assigned to one of three groups: Group A received a referral card for a telephone consultation service; Group B received the same plus a manual on breastfeeding; and Group C received the card, the manual, and a bedside teaching session, which included time to nurse the baby. The telephone follow-up at 30 days postpartum determined that there was a significant difference in breastfeeding rates between the three groups--16% of Group A, 25% of Group B, and 67% of Group C were still breastfeeding.

Of particular interest was a breastfeeding promotion project in Florida which focused on low-income, rural women, many of whom were black (75). The project included individualized instruction, films, and written materials provided in the prenatal clinic, hospital visits twice daily to women who breastfed, discontinuance of formula samples for breastfeeding mothers and early frequent postpartum clinic visits (within a few days after discharge, then weekly for the first month, and monthly thereafter).

Breastfeeding rates before and during the study were compared for 114 women who received prenatal care at the clinic with 373 women who received care from private physicians. The breastfeeding initiation rates for clinic patients were higher than for private patients during each of three six-month intervals after the program was underway (Figure 6). Before the program, no women from the clinic who breastfed continued nursing through 16 weeks postpartum. During the final six months of the program, 48% were still breastfeeding their infants at 16 weeks postpartum.

Similar success has been achieved on several Indian reservations where breastfeeding promotion projects were initiated (75). On the Papago reservation in Arizona, the project involved the development of high quality audiovisual aids to increase community awareness and understanding of breastfeeding. The theme of the material focused on breastfeeding as "the Papago way". Native lay women were recruited, trained and paid as "breastfeeding helpers", both to assist the new mother and infant directly and to act as liaison for her with other services. A free nursery for breastfeeding babies was established in a location central to school and major work places so that new mothers could, with the cooperation of employers, feed their infants during the day. The project also sought to develop rapport with and improve services from the health-care system.

The success of the program is apparent in Table 13 where breastfeeding patterns are summarized. This multilevel approach led to improvements in both initiation and duration of breastfeeding. While the number of mothers initiating breastfeeding has decreased somewhat following the end of the funded program, the percentage of babies breastfeeding during the first months of life remains similar to that during the program. Formula supplementation during breastfeeding has increased. The factors contributing to the apparent post-project declines are not fully understood. However, decreased direct support of the breastfeeding mother may be an influential factor.

A breastfeeding promotion project was also successful on the Fort Peck Indian Reservation in Montana (75). The reservation population is approximately 12,000 and intermarriage with non-Indians and members of other Indian tribes is common. This latter circumstance has led to the loss of many of the traditional ways and breastfeeding has been infrequent for two generations. Few families have contact with childbirth programs although almost all pregnant women receive some prenatal care.

A program of breastfeeding promotion was initiated during the course of prenatal care. Breastfeeding was discussed, literature distributed and a short film shown. The pregnant women were encouraged to talk with other women who they knew had breastfed. The WIC program staff encouraged women "at least to try." The outpatient nursing staff encouraged breastfeeding and the in-hospital routine was made conducive to breastfeeding. Formula samples were not sent home and supplemental feedings were discouraged. Post-hospital discharge follow-up was scheduled within two or three days and generally one week later. Telephone and personal communication was made available. With these promotional efforts, the Fort Peck Reservation demonstrated a percentage of women initiating and continuing breastfeeding among the highest in the state.

It will be interesting to see in coming years how successful breastfeeding promotion programs are in augmenting breastfeeding practices, particularly among disadvantaged blacks and other minorities. In the Ross data, there is little evidence of gain in rates between 1983 to 1984 among poor and minority women. Presumably, breastfeeding rates are plateauing in these subgroups as well as in the population as a whole. But as Faden and Gielen point out (49), whether these rates are plateauing and whether--from the perspective of maternal and child health--this is a cause for concern, are two very important and as yet unanswered questions.

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Table 1.
Causes of maternal mortality in seven states*
participating in a pilot surveillance system, 1983**

	Deaths		Deaths/100,000
Cause	No.	(%)	live births
Embolus	10	(26)	2.5
Peripartum cardiomyopathy	5	(13)	1.2
Cerebrovascular accident	4	(10)	1.0
Hypertensive disease of pregnancy	3	(8)	0.7
Ectopic pregnancy	3	(8)	0.7
Hemorrhage	3	(8)	0.7
Infection	3	(8)	0.7
Anesthesia complications	2	(5)	0.5
Other and unspecified	6	(15)	1.5
TOTAL	39	(100)	9.6

Indiana, Louisiana, Massachusetts, Montana, North Carolina, Oklahoma and Rhode Island

**From Centers for Disease Control, Maternal and mortality: pilot surveillance in seven states. J. Am. Med. Assoc. 255:184, 1986.

Table 2.
Demographic, Physical, Obstetrical and Prenatal Care Characteristics of Women Participating in the Perinatal Collaborative Study*

Marital Status	White	Black
Single	6%	28%
Married	87%	60%
Other	7%	12%
Age (years)		
<17	6.25%	15.92%
18 - 34	85.93%	76.84%
35+	7.81%	7.24%
Education (years completed)		
0 - 8	13.14%	19.00%
9 - 12	65.74%	75.93%
13 or more	21.13%	4.47%
unknown		1.68%
Cigarette Smoking (cigarettes smoked per day)		
0	46.35%	58.15%
1 - 10	23.68%	31.99%
11 - 30	27.15%	9.29%
31 or more	2.83%	0.67%
unknown	1.50%	0.96%
Height (inches)		
under 58	0.99%	1.31%
58 - 66	85.71%	86.83%
67 or more	13.30%	11.86%
unknown	11.04%	3.85%
Prepregant Weight (pounds)		
under 100	5.27%	4.87%
100-119	32.92%	29.84%
120-139	38.49%	34.39%
140-159	14.34%	16.91%
160-179	5.25%	7.51%
180 or more	3.83%	6.50%
Parity		
0	39.99%	34.06%
1 - 2	36.70%	34.12%
3 - 5	19.33%	23.18%
6 or more	3.98%	8.64%
unknown	0.25%	0.24%
Gestation at Registration (weeks)		
0 - 4	0.05%	0.06%
5 - 8	5.25%	1.40%
9 - 16	32.97%	19.99%
17 - 24	27.21%	37.67%
25 - 32	19.33%	29.04%
33 - 36	8.44%	8.47%
37 or more	0.06%	0.13%
unknown		
Number of Prenatal Visits		
1 - 2	6.02%	6.50%
3 - 5	14.32%	23.12%
6 - 8	19.61%	29.31%
9 - 14	48.00%	37.40%
15 - 18	10.71%	3.31%
19 or more	1.34%	0.36%
unknown	0.37%	0.21%

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*Data from Niswander, K.R. and Gordon, M. The Women and Their Pregnancies: The Collaborative Perinatal study of the National Institute of Neurological Disease and Stroke. Philadelphia, W.B. Saunders Co., 1972

Table 3
Maternal Conditions Present During Pregnancy in Women Participating in the Perinatal Collaborative Study*

	White	Black
Organic Heart Disease	1.44%	1.76%
Thrombosis and/or Phlebitis	0.38%	0.17%
Active Tuberculosis	0.05%	0.07%
Pneumonia	0.57%	0.44%
Ashtma	1.30%	2.10%
Diabetes Mellitus	0.66%	0.65%
Hypothyroidism	0.96%	0.32%
Hyperthyroidism	0.22%	0.17%
Glomerulonephritis	0.11%	0.15%
Kidney/Urinary Bladder Infection	10.44%	19.73%
History of Convulsive Disorders	0.55%	0.33%
Convulsions (not eclamptic)	0.35%	0.21%
Mental Retardation	0.41%	0.43%
Neurologic and Psychiatric Conditions	6.00%	2.50%
Cholecystitis	0.23%	0.12%
Cholelithiasis	0.16%	0.06%
Hepatitis	0.09%	0.04%
Appendicitis	0.23%	0.06%
Ileo-colitis	0.17%	0.13%
Peptic Ulcer	0.25%	0.12%

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*Data from: Niswander, K.R. and Gordon, M. The Women and Their Pregnancies: The Collaborative Study of the National Institute of Neurological Disease and Stroke. Philadelphia, W.B. Saunders Co., 1972

Table 4
Complications of Pregnancy and Labor in Women Participating in the Perinatal Collaborative Study*

	White	Black
Hyperemesis Gravidarum	1.64%	0.71%
Pregnancy-Induced Hypertension	5.43%	12.70%
Incompetent Cervix	0.34%	0.36%
Hydramnios	1.54%	1.26%
Placenta Previa (of any degree)	0.77%	0.56%
Abruptio Placenta (any degree)	2.39%	1.90%
Prolapse of the Cord (any degree)	1.10%	0.78%
Uterine Dysfunction (at any stage of labor	8.65%	5.91%
Premature Rupture of Membranes (24 hours before labor or more)	10.79%	21.42%
Puerperal Infection	3.57%	4.14%
Perinatal Death Rate	35.1/1,000 live births	41.9/1,000 live births
Stillbirths	21.8/1,000 live births	22.7/1,000 live births
Neonatal Death Rate	13.6/1,000 live births	19.7/1,000 live births
Offspring Neurologically Abnormal at One Year of Age	17.0/1,000 live	16.0/1,000 live
Birthweight Under 2501 Gm.	71.4/1,000 live births	134.2/1,000 live births

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*Data from: Niswander, K.R. and Gordon, M. The Women and Their Pregnancies: The Collaborative Perinatal Study of the National Institute of Neurological Disease and Stroke. Philadelphia, W.B. Saunders Co., 1972

Table 5
Incidence of low birth weight among racial/ethnic groups in the United States*

	2500 Gm (%)	1500 Gm (%)
White	5.7	0.9
Black	12.5	2.4
Chinese	4.9	0.6
Mexican-American	5.3	0.9
American Indian	6.9	--

*Data from: Behrman, R.E. Preventing low birth weight: a pediatric perspective. J. Pediat. 107:842-854, 1985

Table 6
Incidence of term and preterm low birth weight infants by race, 1970-1980*

Rate per 100 Live Births	Year 1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980
All =											
Low birth weight	7.49	7.09	7.07	7.03	6.94	6.84	6.72	6.51	6.51	6.37	6.31
Term low birth weight	3.58	3.29	3.24	3.30	3.32	3.18	3.06	2.93	2.91	2.79	2.76
Preterm low birth weight	3.81	3.80	3.82	3.71	3.62	3.66	3.66	3.59	3.60	3.58	3.55
White											
Low birth weight	6.44	6.18	6.06	6.00	5.93	5.85	5.73	5.52	5.49	5.37	5.32
Term low birth weight	3.20	2.94	2.82	2.85	2.87	2.74	2.64	2.53	2.49	2.37	2.35
Preterm low birth weight	3.24	3.23	3.23	3.15	3.05	3.11	3.09	3.00	3.00	3.00	2.97
Black											
Low birth weight	12.97	12.54	12.69	12.54	12.45	12.17	12.01	11.81	11.93	11.70	11.70
Term low birth weight	5.77	5.46	5.53	5.73	5.72	5.48	5.27	5.04	5.08	4.97	4.92
Preterm low birth weight	7.20	7.08	7.15	6.81	6.73	6.69	6.73	6.78	6.84	6.73	6.77

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*Rates calculated from data provided by the National Center for Health Statistics for states reporting birth weight and date of last menstrual period for each year. From: Kessel, S.S., Villar, J., Berendes, H.W., and Nugent, R.P. The changing pattern of low birth weight in the United States JAMA 251:1978-1982, 1984

=Low birth weight indicates a birth weight of 2,500 g or less; term, at least 37 completed weeks of gestation; preterm, under 37 completed weeks of gestation.

Table 7

Incidence of low birth weight in relation to maternal education

Maternal education (yr)	Relative risk						Total live births (1981) (%)	
	White			Black				
	1971	1976	1981	1971	1976	1981	White	Black
0 to 8	1.35	1.39	1.46	1.08	1.14	1.17	3.7	4.7
9 to 11	1.32	1.35	1.47	1.10	1.12	1.18	15.8	30.1
12	0.92	0.94	0.96	0.92	0.92	0.95	44.0	41.5
13 to 15	0.80	0.82	0.81	0.81	0.85	0.83	18.9	15.5
≥ 16	0.74	0.73	0.74	0.74	0.72	0.71	16.2	6.5

*Includes 49 states and Washington, D.C. in 1981; 44 states and Washington, D.C. in 1976; 38 states and Washington, D.C. in 1971.

Data derived from: Behrman, R.W. Preventing low birth weight: a pediatric perspective. J. Pediatr 107:842-854, 1985.

Table 8

Estimated number of births of all live infants and of low-birthweight infants, by region, 1982, and estimated proportion of low-birthweight infants, 1979 and 1982

Region	Live Births*	Low-birth-weight infants		
			Percentage=	
		1982*	1979	1982
Africa	23,148	3,233	15.0	14.0
North Africa	4,814	495	13	10
Western Africa	7,278	1,256	17	17
Eastern Africa	6,930	922	14	13
Middle Africa	2,554	398	15	16
Southern Africa	1,372	162	15	12
North America	4,402	299	7.3	6.8
Latin America	12,490	1,259	10.2	10.1
Middle America	3,669	448	12	12
Caribbean	867	102	13	12
Tropical South America	7,033	647	9	9
Temperate South America	921	62	8	7
Asia	74,885	14,750	20.3	19.7
Western South Asia	4,080	302	16	7
Middle South Asia	35,311	10,947	31	31
Eastern South Asia	12,336	2,088	18	17
East Asia	23,158	1,413	6	6
Europe	6,857	445	7.7	6.5
Northern Europe	1,010	61	6	6
Western Europe	1,819	95	6	5
Eastern Europe	1,855	140	8	8
Southern Europe	2,173	149	9	7
Oceania	507	59	12.2	11.6
Union of Soviet Socialist Republics	5,111	409	8.0	8.0
World	127,400	20,450	16.8	16.0
Developed countries	18,200	1,250	7.4	6.9
Developing countries	109,200	19,200	18.4	17.6

*In thousands.

=Decimals are only shown for continents, since estimates for subregions are subject to a greater margin of error.

Previous estimate for Middle America corrected.

Sources: United Nations, Department of International Economic and Social Affairs. Demographic indicators of countries: estimates and projects assessed in 1980. New York, 1982.

Table 9
Risk factors associated with adverse pregnancy outcome

I. Demographic risks
   - A. Age (younger than 18, older than 35 years)
   - B. Race (black)
   - C. Low socioeconomic status
   - D. Unmarried
   - E. Low level of education

II. Medical risks predating pregnancy
   - A. Parity (0 or more than 4)
   - B. Low weight for height
   - C. Genitourinary anomalies/surgery
   - D. Selected diseases such as diabetes, chronic hypertension
   - E. Nonimmune status for selected infections such as rubella
   - F. Poor obstetric history including previous RBW infant, multiple spontaneous abortions
   - G. Maternal genetic factors

III. Behavioral and environmental risks
   - A. Smoking
   - B. Poor nutritional status
   - C. Alcohol and other substance abuse
   - D. Diethylstilbestrol and other toxic exposures, including occupational hazards
   - E. High altitude

IV. Health care risks
   - A. Absent or inadequate prenatal care
   - B. Iatrogenic prematurity

V. Medical risks in current
   - A. Multiple pregnancy
   - B. Poor weight gain
   - C. Short interpregnancy interval
   - D. Hypotension
   - E. Hypertension/preeclampsia/toxemia
   - F. Selected infections such as symptomatic bacteriuria, rubella, cytomegalovirus
   - G. First- or second-trimester bleeding
   - H. Placental problems such as placenta previa, abruptio placentae
   - I. Hyperemesis
   - J. Oligohydramnios/polyhydramnios
   - K. Anemia/abnormal hemoglobin
   - L. Isoimmunization
   - M. Fetal anomalies
   - N. Incompetent cervix
   - O. Spontaneous premature rupture of membranes

VI. Evolving concepts of risk

- A. Stress (physical and psychosocial)
- B. Uterine irritability
- C. Events triggering uterine contractions
- D. Cervical changes detected before onset of labor
- E. Selected infections such as mycoplasma and Chlamydia trachomatis
- F. Inadequate plasma volume expansion
- G. Progesterone deficiency

Table 10

Percentage of Infants Breastfeeding in Hospital and at 5 and 6 months by Selected Demographic Characteristics—Revised Weights—Ross Laboratories Mothers Surveys

	In hospital			At 5 and 6 months		1985=
	1983*	1984*	1985	1983*	1984	(at 5 mo)
Ethnicity						
White	64.4	65.0	64.3	28.9	28.6	29.0
Black	29.8	33.3	30.0	9.7	11.7	10.8
Maternal age						
Less than 20 yr	39.3	36.8	37.3	8.7	10.2	9.0
20-24 hr	55.5	58.0	55.2	19.1	19.7	20.1
25-29	65.3	66.6	64.9	31.1	30.6	30.3
30-34	67.8	69.4	70.1	38.0	39.1	39.1
35 yrs or older	60.7	64.7	62.8	34.9	38.5	38.8
Family income						
Less than $7,000	36.2	36.6	NA	12.3	12.4	NA
$7,000-14,999	54.0	54.5	NA	20.4	21.1	NA
$15,000-24,999	63.8	64.8	NA	29.2	29.2	NA
$25,000 and over	71.3	71.8	NA	33.9	33.4	NA
Maternal education						
Noncollege	49.1	50.7	NA	17.8	19.2	NA
College	76.7	77.5	76.4	39.8	38.8	39.5
Maternal employment						
Employed full time	55.5	56.2	56.2	12.2	12.3	12.9
Employed part time	65.9	67.6	63.8	28.6	28.9	28.4
Total						
Employed	59.3	60.3	NA	17.5	18.2	NA
Not employed	58.0	58.6	57.9	29.0	29.2	29.9
U.S. census region						
New England	57.8	58.5	60.8	27.4	25.8	28.4
Middle Atlantic	48.9	50.9	49.6	23.1	23.4	23.1
East North Central	57.1	57.7	55.8	24.1	24.9	24.8
West North Central	62.3	66.0	65.6	26.9	28.2	27.7
South Atlantic	50.2	50.5	50.4	20.2	20.2	20.4
East South Central	42.7	45.9	44.5	16.1	17.3	17.6
West South Central	55.4	55.9	51.5	20.0	20.2	19.0
Mountain	75.8	77.6	76.8	36.4	37.6	37.4
Pacific	77.4	77.7	76.1	34.8	36.0	36.0
WIC						
Yes	NA	NA	40.9	NA	NA	14.3
No	NA	NA	66.1	NA	NA	30.7

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*Reported by permission of Martinez GA, Krieger FW. 1984 Milk feeding patterns in the United States. Pediatrics 1985;76:1004-8.

=Derived from Martinez GA, Stahl D. Geographic and demographic differences in infant feeding. Presented at the APHA Annual Meeting. Washington, D.C.: November 20, 1985, 1985 data are preliminary through the 3rd quarter of the year.

NA = data not available at this time.

Table 11
Demographic Characteristics of University of Texas
1981 Sample (n=358)

Women Characteristics	No.	breast-feeding (%)	$/X^2$ (df)	p*
Marital status				
Married	218	35.8	20.66 (2)	<0.0001
Single	93	14.0		
Other (widowed divorced	45	13.3		
Head of household				
Husband	200	36.0	18.52 (2)	<0.0001
Self	56	19.6		
Other	101	13.9		
Pregnancy				
1st	123	35.0	6.02 (2)	0.0492
2nd	118	24.6		
3rd or more	116	21.6		
Maternal ethnicity				
Anglo-American	145	43.5	43.69 (3)	<0.0001
Black American	131	9.2		
Mexican American	62	22.6		
Other	19	42.1		
Paternal ethnicity				
Anglo-American	133	44.4	53.63 (3)	0.0001
Black American	138	9.4		
Mexican American	63	19.1		
Other	21	57.1		
Maternal education				
Grade 8 or less	68	29.4	10.64 (3)	0.0138
Some high school	117	18.0		
Graduate of high and or technical school	101	28.4		
At least some college	68	39.7		
Annual family income**				
<$5,000	61	18.0	9.74 (2)	0.0077
$5,000-15,000	75	33.3		
>15,000	38	47.4		

Reproduced by permission of Rassin DK, Richardson J, Baranowski T, et al. Incidence by breast-feeding in a low socioeconomic group of mothers in the United States. Pediatrics 1984;73:134.

*Observed significance level of the X^2 test for equality of proportion across the indicated categories.

**Of the total sample, 184 either did not know annual family income or did not respond.

Table 12
Breast-feeding by Demographic Characteristics WIC—1984 Ross Laboratories Mothers' Surveys

		Breast-fed (%)	WIC births
1	Pacific	69	11
2	Mountain	61	5
3	College	60	13
4	$15,000 + income	52	16
5	West North Central	50	6
6	$10,000-$14,999	48	19
7	White	48	60
8	30-34 yr	46	7
9	35+ yrs	46	2
10	25-29 yr	45	18
11	20-24 yr	44	40
12	Employed	42	24
13	Hispanic	42	12
14	Multiparous	41	48
15	New England	40	5
16	East North Central	39	20
17	Primiparous	38	52
18	Not employed	37	76
19	No college	36	87
20	<$10,000 income	35	65
21	West South Central	34	10
22	Middle Atlantic	33	17
23	Premature babies	32	8
24	<20 yr	28	33
25	South Atlantic	26	16
26	East South Central	25	10
27	Black	25	34
	National	39	

Reproduced with permission from Martinez GA, Director of Market Research.

Table 13

Breastfeeding Initiation and Duration
(Papago Reservation—1979 through 1983)*

	1979	6/81	7/81-6/82	4/83-9/83
Percentage of New Mothers Initiating Breastfeeding	23	44	59	49
Percentage Breastfeeding at				
6 weeks			50	48
4 months			33	42
Percentage Utilizing Formula Supplementation (Age: Birth to 4 months)			21	37

* From: Alden, J.E. Breastfeeding promotion in three rural indigent populations. In: Report of the Surgeon General's Workshop on Breastfeeding and Human Lactation. USDHHS Pub No HRS-D-MC 84-2, Washington, D.C., 1984, p.44

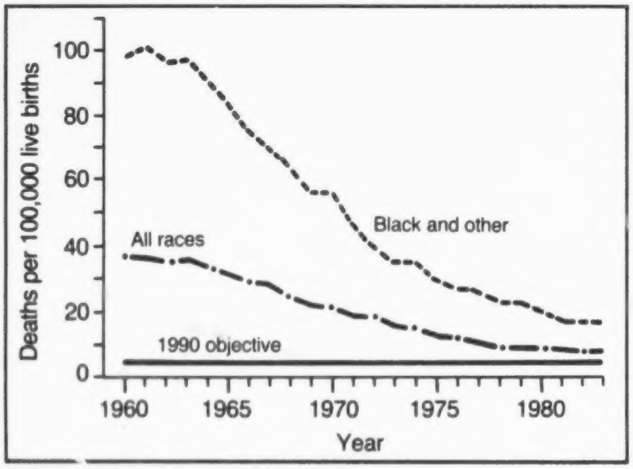

Figure 1: Maternal Mortality rates, by race, United States 1960-83. From: Centers for Disease Control, Maternal mortality: pilot surveillance in seven states. JAMA 255: 184-185, 1986

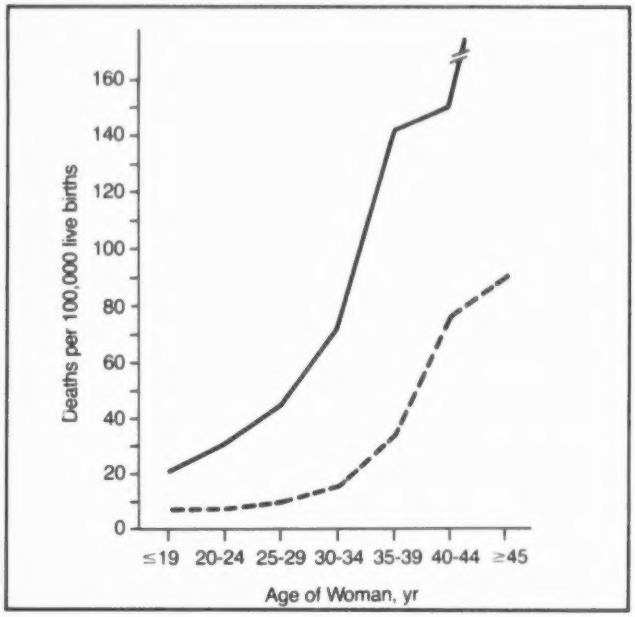

Figure 2: Maternal mortality rate by age of woman, United States 1974 through 1978. Solid line indicates black women; dashed line, white women. From: Buehler, J.W. , Kaunitz, A.M. Hogue, C. J. R., Hughes, J.M., Smith, J.C., and Rochat, R.W. Maternal mortality in women aged 35 years or older: United States. JAMA 255: 53-57, 1986

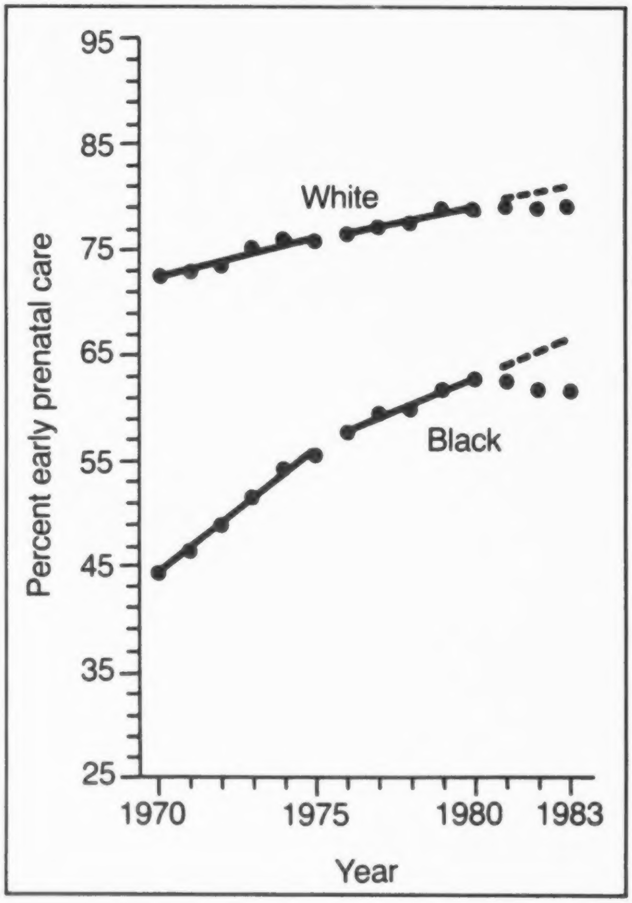

Figure 3: Observed and fitted percent of mothers with early prenatal care, by race, 1970-1983. (Note: Percents based on births to residents in the 40 states with prenatal care data for the entire period, 1970-1983) From: Ingram, D.D., Makuc, D., and Kleinman, J.C. National and state trends in use of prenatal care, 1970-83. Am. J. Pub. Health 76: 415-423, 1986

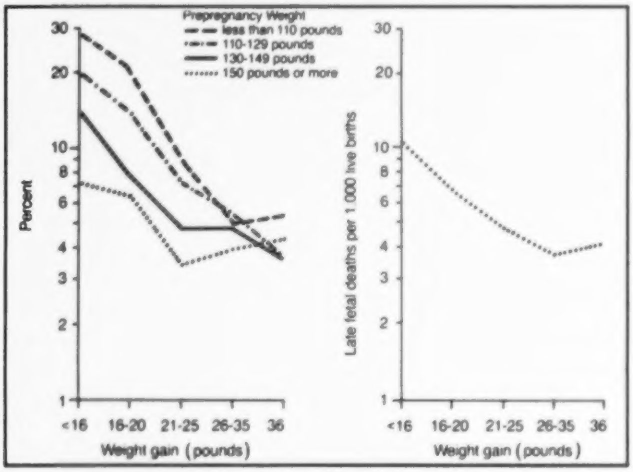

Figure 4: Percent of liveborn infants of low birth weight by maternal weight gain during pregnancy, according to mother's prepregnancy weight: United States, 1980 National Natality Survey. B. Fetal death ratios by weight gain during pregnancy: United States. 1980 National Natality and Fetal Mortality Surveys.

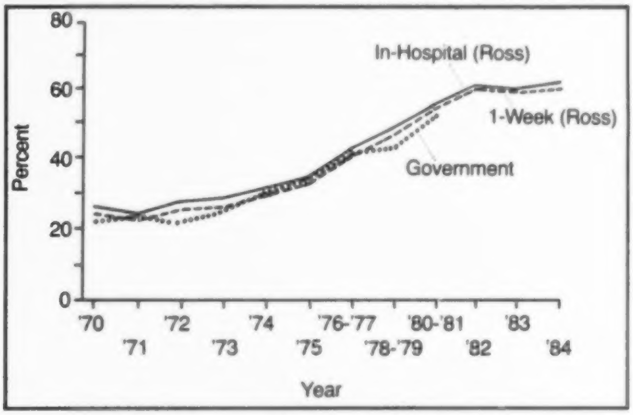

Figure 5: Incidence of breast-feeding U.S.A. Percent of infants breastfed by years 1970-83. Sources: Ross Laboratories National Mothers Survey and National Survey of Family Growth, National Center for Health Statistics. (Reproduced by permission from Martinez, G.A., Krieger, F.W. 1984 Milk feeding patterns in the United States. Pediatrics feeding patterns in the United States. Pediatrics 76:1004-1008, 1985)

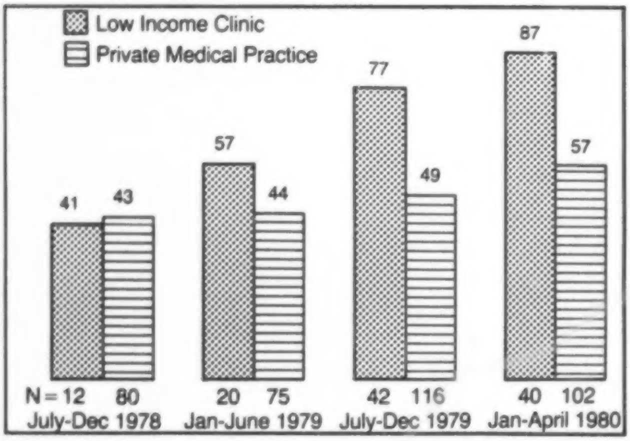

Figure 6: Percentage of women initiating breastfeeding of newborns delivered by private physicians vs. those cared for in low income clinic. Subjects derived from an indigent rural county in Florida. From: Alden, J.E. Breastfeeding promotion in three rural indigent populations. In: Report on the Surgeon General's Workshop on Breastfeeding and Human Lactation. USDHHS, Washington, DC., 1984, p.41

Nutrition in the Infant and Child

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Introduction

The previous speakers have addressed the problem of the extent and severity of undernutrition in the United States. They have shown that undernutrition is concentrated among blacks and other minorities. They have implied, at least, that the major sufferers among those groups are infants and children. We have also heard that certain diet-related chronic diseases such as obesity, hypertension, atherosclerosis and cancer are too common among Americans and again are more prevalent among blacks and minorities. I would like to discuss what the implications for childhood nutrition are. Thirdly, mention has been made of the epidemic of teenage pregnancy, again showing a preponderance among blacks and minorities. The results of such an epidemic are low birth weight, poor neonatal care, poor environment for both mother and infant, and a cycle which repeats itself over and over again. From here on we are going to discuss the health consequences of these very severe problems. Therefore, I would like to address four very specific questions. First, do hunger and malnutrition affect the biological and functional development of the brain? Second, do feeding patterns as applied to infants in the United States contribute to further health in terms of infant growth, infection and subsequent disease, and do these feeding patterns impact particularly on blacks and minorities? Third, is the pattern of feeding children contributing to such diseases in adults as obesity, hypertension and atherosclerosis? And fourth, what are the health risks of adolescent pregnancy and where does poor nutrition fit into these risks?

I. Undernutrition and the Developing Central Nervous System

During the 1960's and early 1970's a great deal of evidence was accumulated in animals which demonstrated that undernutrition, if imposed at critical periods when cells are actively dividing in the central nervous system (CNS), could impair the rate of cell division and permanently stunt the brain by producing a brain with fewer cells. In the 1970's the same kinds of data were gathered for humans. Human brain weight increases linearly from before birth until beyond one year of age. By contrast, the number of cells as measured by the DNA content of the brain increases linearly until birth, then begins to level off and by 18 months of age we have all the cells in our brain that we are going to have. So if one is interested in the effects of early malnutrition on cellularity of the brain from the standpoint of impeding the rate of cell division, then one is interested in that kind of malnutrition which occurs in the first 18 months or one year of life. This type of undernutrition is called infantile marasmus. Among other things it causes stunting and a marked reduction in head circumference. Even today, about 20 percent of children who present with this problem will die. Actual analysis of brains from such children has been carried out. In the other 80 percent the child can be placed in a program which prevents malnutrition and the outcome, in terms of mental development, determined. If you analyze the brains of such children, there is a reduced number of cells in the cerebrum, cerebellum and brain stem. This reduction in cell number is also accompanied by a reduction in myelination, as measured by a reduced total cholesterol content and a reduced phospholipid content in the brain. All of these changes had been shown in animals to be permanent changes.

If these children should survive, a number of studies have shown that behavioral abnormalities will occur. These can include reduced IQ scores and poor school performance. Thus by the end of the 1970's, there was real concern from human data that biological damage can be done to the CNS by severe undernutrition and that such biological damage could manifest itself in behavioral abnormalities. The 1980's showed that the problem was even more complicated because what we have learned is that it is not simply undernutrition which is causing the behavioral changes. Rather, the behavioral changes are a function of malnutrition in a particular type of environment - an environment which is somehow isolating this child from the normal stimuli that a child of this age should be getting. Two studies really focused our attention on this. One was a study in children with cystic fibrosis who had been every bit as malnourished as the children from the third world I have been describing. When such children were examined, by age 6 they showed perfectly normal IQ scores and normal behavioral patterns. What was different about the cystic fibrotic child when compared to the typical poverty child who suffers the same amount of malnutrition? One difference was that the child with cystic fibrosis was getting major amounts of stimulation whereas the child born in poverty was not. A study was done to test the effect of stimulation in malnourished children. Three groups of Korean children were examined. One group had been severely malnourished during the first six months of life (below the 3rd percentile for height and weight). One group was moderately malnourished (below the 25th percentile for height and weight), and the last group was well nourished (at or above the 50th percentile). All of these children were adopted by middle class U.S. families before the age of three. By age 12-15, the average IQ of the previously malnourished group was 102, right at the U.S. norm. For both IQ and school performance the malnourished groups were normal whereas the well nourished groups were achieving even better than the U.S. norms. If the intervention (i.e., the stimulation) occurs after age 3, everybody's IQ drops and the malnourished children do significantly worse than the U.S. norms. The later the stimulation, the worse the result. Thus, early stimulation combined with good nutrition seems to be able, at least in part, to prevent the expected behavioral effects. Some studies suggest, although they do not demonstrate, that the same types of forces may be operating in the United States. Assuming that a child who is shuttled between foster homes is in an unstable environment, whereas a child who remains in one foster home is in a relatively stable environment, the two situations can be compared from the standpoint of IQ. The children in the multiple foster home experience have an IQ 14 points lower than those raised in a single foster home. Certainly, it is quite possible that the mothers who get the child with the low IQ may send that child off. Therefore, which is cause and which is effect? However, if one other variable is examined the situation becomes more clear. Malnourished children are more likely to be stunted and if we plot the height of the children in the single foster home group against their IQ scores, you can see that there is no correlation. Thus, it made no difference whether or not they were tall or whether they were short in terms of what their IQ would be. By contrast, if they were raised in multiple foster homes, the correlation was quite good. It was the short children who were apt to have the lower IQ's, suggesting an interaction between something which has resulted in short stature and subsequent environment.

The questions raised by some of the data I have just discussed are as usual greater than the questions answered, but I think we can conclude certain things.

1. Severe early malnutrition during at least the first year to 18 months of age will produce structural changes in human brain as reflected by a small head circumference, a reduced number of cells, abnormal and insufficient myelination, poor dendritic arborization and poor formation of gangliosides.

2. Such early undernutrition, when combined with poor environmental stimulation, will lead to a reduction in the function of the brain as measured by IQ scores, school performance and by a host of other behavioral abnormalities.

II. Feeding Patterns in Early Infancy and Future Health

As we have seen, breast-feeding is less frequent among blacks and minorities. There is no doubt that breast-feeding is important. The data are very clear, not only from the standpoint of the nutritional aspects of breast-feeding, but from the probably much more important immunological aspects of breast-feeding. The breast-fed infant has an enormous immunological advantage, particularly during the first 6 months to a year of life. This is particularly related to two categories of disease, gastrointestinal disease and upper respiratory disease. A previous report mentioned that the infection rate among American Indian babies was high. The incidence of breast-feeding varied between 27% in some reservations to about 12% in other reservations. I wonder whether or not there is an association between the low breast-feeding rates and the high infection rates? But, there is another thing about breast-feeding which is very important. There is good evidence that bottle fed babies gain more weight during the first year of life than breast-fed babies.

Some evidence suggests that there may be a relationship between childhood obesity and too rapid weight gain during the first few yeas of life. In addition, mothers who bottle feed will introduce solid foods earlier than mothers who breast-feed and the early introduction of solid foods is usually associated with a caloric intake well above the requirement of the infant. Again, we worry about this from the standpoint of obesity and therefore to the extent that obesity is a problem of greater magnitude among blacks than among whites. It becomes a problem that blacks especially have to worry about. Lastly, because of a unique mechanism for binding iron within breast milk, the breast-fed infant is able to absorb up to 50% of the maternal iron that is in breast milk, higher than from any other food. For this reason breast-fed infants do not get iron deficiency. The early cessation of breast-feeding can lead to iron deficiency particularly if formulas are made up that do not contain iron or if the mother is in a hurry to introduce whole milk which does not contain iron. Thus iron deficiency, also a problem of greater magnitude among blacks than among whites, is a problem that may be magnified by the lack of breast-feeding in that population.

III. Childhood Antecedents of Adult Disease

A question that is being asked more and more is, "Are there ways in which we are feeding our children which are leading to the pattern of adult chronic disease that we see later on?" Three diseases have been focused upon - obesity, hypertension and atherosclerosis.

First, does obesity in childhood lead to obesity in adults? The answer is very clear. Yes. If a child is obese by three years of age, such a child has better than a 70% chance of being an obese adult. Obesity in childhood therefore becomes a problem which is important for the general population but particulary important to the black population and to other minority populations in which obesity occurs at higher incidence. That is not to say that if you are thin at age 3 and get obese in adolescence you are not going to be an obese adult. You are, but obesity in childhood is a very specific problem. And we know today that obesity in childhood results in a specific form of obesity, the so-called hyperplastic obesity, in which there is an increased number of fat cells. This type of obesity is very recalcitrant to treatment later on.

The focus on hypertension has not been as great, yet the need is probably as great. There is no question that the earlier an individual develops hypertension, the more he or she is at risk for the complications of hypertension. So, the detection of hypertension early in life is an extremely important responsibility of anybody taking care of children. The relation of salt to blood pressure is discussed else where. There are very strong data that suggest that salty is a learned taste in comparison to sweetness, which is an inborn taste.

Infants are born favoring sweet solutions, they are not born favoring salty solutions. They learn that, and to the extent that we can prevent them from learning that, we may be making the job of preventing hypertension later in life much easier. The pattern of introducing solid foods, particularly among blacks and minorities, should encourage the use of low-sodium products, those which are not highly processed and which do not contain added salt. Because of the possible risk for atherosclerosis, a consensus conference of the NIH recommended that every child under the age of two should be on a low fat diet (30% of calories or less). The American Academy of Pediatrics Nutrition Committee responded that there are no data that such a diet is going to be doing any good and potentially it could be doing harm. What about a child who has previously undergone a severe bout of malnutrition, consuming a diet which is particularly low in fat? Although I am not concerned with the recommendation of 30% of calories or less, I am concerned with the mother who says, "Well, if my child will do well on 30% of calories as fat, let's give him 10%." And I am even more concerned about when you translate this to food, because it means using skimmmed milk and skimmed milk products. Two year olds still get a large amount of their caloric intake from milk and if one uses skim milk and skim milk products, expense is increased without knowing if any benefit will occur.

IV. Adolescent Pregnancy

We have heard that adolescent pregnancy has reached epidemic proportions. We have also heard that it is also more prevalent among blacks and among minorities. Both the infant and the young adolescent are at risk. We have heard about the importance of weight gain during pregnancy and it was shown that 26 to 35 pounds would give you the best size baby. But, that is an average for well nourished, adult population. No adolescent fits into that population. Dr. Pedro Rosso has come up with another way of assessing how much weight a woman should gain. He has shown that the best chance for a woman to have a normal size baby is for her to reach 120% of her ideal pre-pregnancy weight at term. Thus, not only how much weight you gain is important, but your nutritional status before you became pregnant is also important. Using this concept, the adolescent becomes a very high risk patient. If the adolescent is under weight, she is going to have to make that weight up during pregnancy. In addition, she is also going to have to gain enough weight to cover the amount of weight she would have gained during this period if she were not pregnant. Some adolescents may need to gain 50 or 60 pounds. Since the adolescent is generally smaller, the complications of an adolescent gaining 50 or 60 pounds during pregnancy can be considerable. Finally, adolescents for a number of reasons - not the least of which is that they may wish to hide the fact that they are pregnant - want to gain as little weight as possible. Thus, most pregnant adolescents do not gain enough weight to insure the best chance for a normal sized infant. It is no surprise therefore, that the birth weight in adolescent pregnancy is significantly below the birth weight in non-adolescent pregnancies in both blacks and whites. The black adolescent has a much worse outcome than the black non-adolescent, even though both are more likely to have a low birth weight infant. Finally, the infant enters the vicious cycle of a low birth weight infant who then goes into a poor environment and gets poor nutrition.

In summary, early malnutrition can affect the biologic and functional development of the brain when it occurs in a poor home environment. Lack of breast-feeding and early beginning of solid foods contribute to a pattern which may increase the incidence both of iron deficiency and of subsequent obesity. Childhood obesity and hypertension are very serious antecedents of future chronic diseases, and the problems which may have particular relevance to blacks. And finally, pregnant adolescents are at special nutritional risk and their infants enter a vicious cycle from which it is very difficult to withdraw.

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Nutrition and Aging

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Introduction

The impact of age on health and nutritional status is being noticed by more health care professionals as the elderly comprise an ever increasing segment of the American population. In 1900, there were approximately 3 million Americans over age 65 and by 1980 there were almost 25 million in this age group. It is projected that by the year 2040, there will be 55 million people over age 65 (1). Part of the reason for this increase is the increasing number of people who are living longer, well into their 80s and 90s.

This shift in demographics is important, not only because of the large number of people that it entails, but also because this segment of the population includes the greatest consumers of health care resources in the United States, using 40% of acute care hospital stays, purchasing 25% of all drug prescriptions written, and accounting for more than 50% of the federal health care budget (2). The economic impact of chronic illnesses, which are frequently found in the older population, on the prospective payment system, the utilization of acute care beds and the search for alternative services and facilities for elderly patients who require lengthier recuperative and rehabilitative care, has yet to be clearly defined.

Physiologic Aging

It is unknown whether there are nutritional factors that impact on the rate and manner in which individuals age. To conduct valid longitudinal studies takes several human lifetimes and ongoing teams of researchers. Presently, the Baltimore Longitudinal Study on Aging (BLSA) is the premier example of a study on human aging that is attempting to address the enormous problems associated with this type of research (3). Thus far, the BLSA has been able to document changes that are associated with normal physiologic aging, but there are little data on the role of nutrition in this process. However, data from this study have provided scientists and medical practitioners with information that demonstrates how normal aging impacts on nutritional requirements and status.

Aging is a slow, inexorable process that occurs in all living organisms. The factors that control aging are difficult to quantitate. They include various life events; illnesses, both acute and chronic, stress, socioeconomic status, access to health care through life, the environment (climate, living conditions, acts of nature such as fire, flood, earthquake and tornado); and genetic heritage. All of these factors contribute to the rate and manner in which people age. There is enormous variability between individuals, even within the same family, and there is variation in the rate of aging among organ systems within an individual (4). It is, therefore, very risky to make generalizations about human aging.

There are changes that occur universally and can be associated with aging in all humans. There is a loss of cells from active, functioning tissue.

Protein tissue is the most active metabolic tissue in living organisms, and the loss of cells is primarily from tissue protein, from muscle and visceral compartments. Muscle tissue losses will affect skeletal function as well as smooth muscle action, such as cardiac function. Lungs, kidney and liver tissues will also be lost with an associated compromise in function. Organs that are not affected by the presence of chronic or acute disease will function normally but with age, reserve capacity, or the ability to respond to stress, will diminish.

Another consequence of the loss of protein and cells is a loss in immune function. The normal reaction to aberrant cell production or invading microorganisms is slow or inadequate. Therefore problems with primary cancers or infection may be more prevalent in older age groups, which indeed is the case (5).

One obvious outcome that is associated with the reduction in total body protein is a lowering of basal metabolic rate. Since protein tissue is the most metabolically active tissue, the decrease in its total contribution to body mass must be associated with lower basal metabolism. This translates into a decreased requirement for dietary energy sources to maintain body weight at a constant level.

Other change, associated with a loss of lean body mass, is the loss of total body water. Since total body water is primarily located in tissue protein as interstitial water, as lean body mass decreases, body water will decrease proportionately. Additionally, total body fat increases.

The other major change in body composition is a loss of bone density. This occurs in all humans as they age and the loss of bone density is called osteoporosis when it reaches the pathological stage.

These physiological changes are manifested by functional changes in organ systems. Functions of organ systems will diminish over time leading to a decrease in functional parameters such as cardiac index, stroke volume, vital capacity, maximal breathing capacity, standard glomerular filtration rate, standard renal plasma flow, and basal metabolic rate (3). Other age related changes that impact on nutritional status include the ability to obtain adequate dietary intake. For example, taste and smell sensitivities have been reported to change with increasing age, thus affecting the appeal of many foods. Food that has no discernible taste or odor will not be eaten. Digestive enzyme secretion decreases over time, contributing to the indigestibility of many foods (3). Real or perceived food intolerance will lead to the elimination of many foods from the diet. Decreasing variety makes eating less pleasurable, and appetite diminishes accordingly. If elderly people are not interested in their food, they will not make an effort to eat more than they need to assuage their hunger. Although this may contribute to maintaining weight, it will make meeting nutrient requirements more difficult, and may lead to weight loss.

An important obstacle to ingesting an adequate diet is the decrease in dentition that occurs with advancing age. Many of the older population have lost teeth due to poor dental care and lack of knowledge about dental hygiene when they were young. Many have dentures, but due to loss of lean body mass (gum tissue) and decreased bone density (changes in jaw bones), dentures do not fit well and are therefore not worn. For many people, their inability to adequately chew certain foods leads to the latter's elimination from the diets. This contributes to further limitations for many because the foods most often left out are meats, fresh fruit and vegetables. This will obviously impact on the nutritional quality of their dietary intake.

Environmental factors

In addition to the changes that occur with normal aging, older people have to cope with a changing environment. Children have grown up and left home, spouses and old friends die, leaving elderly people without companions. For elderly men in particular, this may prove extremely difficult. They do not have food procurement preparation skills, and have been shown to be at greater risk for nutritional problems than are elderly women.

Many elderly people face the prospect of living on fixed incomes in an increasingly expensive world. Fresh meats, fruits and vegetables are expensive foods, and may be eliminated from the diets due to economic concerns, particularly during bad weather seasons of the year when going shopping for fresh food may be difficult. The elderly may also suffer from chronic illnesses which may limit their independence, interfere with their mobility and eventually lead to institutionalization. This will impact on their attitudes and outlook on the world and contribute to loss of self-esteem, a feeling of increasing isolation, loss of motivation to care for themselves, and ultimately severe depression. When working with the elderly, it is important to consider their lifestyles, recognize that they have varied and complex histories, and that they have seemingly overwhelming problems to cope with. Nutrition frequently becomes a low priority.

Factors that interfere with adequate intake

There are a multitude of factors that interfere with the ability of some elderly people to have an adequate dietary intake. Several of them have been discussed already. They include environment as well as psychological and sensory changes associated with aging. Taste and smell acuity losses have been mentioned, but decreases in visual, auditory and tactile senses will also contribute to nutritional risk. Impairment of vision will restrict mobility, make it difficult to read labels, prices, and recipes, and limit access to nutrition education materials. Hearing losses make communication with the outside world difficult and lead to reluctance to ask questions and collect information. Tactile sensitivity losses, along with arthritis and decreasing fine motor skills will make food preparation less appealing.

Intercurrent illnesses, such as cardiac disease, diabetes, osteoporosis, osteomalacia, chronic gastrointestinal conditions, urinary tract infections, and others may all impact on nutritional status. Some of these conditions may alter nutritional requirements for a brief time and others may have longer term effects. Changes in diet are difficult for elderly patients to make. Their comprehension of nutritional principles and their willingness to change lifetime eating habits may be limited. Modifying diets to manage chronic diseases may not be successful, and may serve to further impair dietary intake.

Associated with intercurrent illnesses is the use of multiple prescription and over-the-counter medications. Many prescription drugs will interfere with nutrient absorption or utilization (6). Drug profiles should be developed to avoid potential nutritional deficiencies associated with polypharmacy in the elderly. It is very important to obtain a thorough drug history from elderly people. They may be on medications prescribed by many different physicians who are unaware of what each other has given to the patient. In addition, elderly people may also be taking multiple over-the-counter drugs and remedies, which may interfere with appetite, nutrient absorption and utilization.

Nutritional requirements

Nutrient requirements have not been clearly defined for people aged 65 and over. Perhaps one of the reasons for this is the variety and heterogeneity in this age group. The older people are, the more unique each individual becomes. Nutrient requirements can be affected by so many variables that coming up with specific levels for all the nutrients is rather difficult (7). We do know that requirements for calories decrease over time (3). As previously discussed, due to less metabolically active tissue, basal metabolic rate decreases contributing to a decreased need for energy. Also, activity levels tend to decrease with advancing age, making the need for calories lower. Unfortunately, the need for other essential nutrients does not decrease as does calories. Obtaining adequate amounts of all the other nutrients despite decreased calorie requirements is challenging.

Protein, as a major macronutrient, is the object of considerable research. There is no definitive evidence that protein needs change with age (8). Obtaining adequate dietary protein may be difficult for some members of the older age groups due to the expense of high protein foods. Economic restrictions may lead some elderly to avoid these foods and some elderly may be on protein-restricted diets because of chronic renal or hepatic disease. For these people, it may be difficult to balance therapeutic restrictions with adequate protein intake to prevent protein-energy malnutrition from developing.

Carbohydrates constitute the greatest percent of calories in most diets. Incorporating complex carbohydrates, particularly from fresh fruits and vegetables and whole grain cereal products is a reasonable goal. These foods, however, are often not included in diets of elderly people. It is important for health care professionals to encourage use of these carbohydrate sources for several reasons. Glucose tolerance tends to decrease with advancing age so that using simple carbohydrates may stress renal glucose thresholds and increase already increased circulating levels of glucose (9). Using complex carbohydrates also adds fiber to the diet. Increased amounts of dietary fiber will promote greater bowel motility and contribute to correcting chronic constipation.

Fat is another macronutrient that must be considered. Fat contributes fat soluble vitamins and calories. However, fat soluble vitamins can often be obtained from water-soluble sources (e.g., beta-carotene available from many vegetables is a precursor for vitamin A; milk is supplemented with vitamin D; vitamins K and E are found in whole grain cereals). Fat can be used in limited quantities to control total caloric intake without jeopardizing the overall nutrient content of the diet.

Often forgotten as a nutrient, water is very important to monitor in elderly people, especially those who are chronically ill or institutionalized. Osmoreceptors decrease in sensitivity with age and thirst responses decrease over time, making the elderly less responsive to the physiologic need for water than younger people. Elderly people are sensitive to shifts in fluid compartments, and rapid dehydration can contribute to a variety of potentially serious problems. Symptomatology may include hypotension, elevated body temperature, constipation, nausea, vomiting, mucosal dryness, decreased urinary output, and mental confusion. The signs of dehydration, dryness of the skin and mucosa, sunken eyeballs, loss of skin turgor, constipation and confusion, among others, are often ascribed to old age and not recognized as symptoms of a correctable problem (10). Adequate fluid intake must be encouraged in this population. Requirements for water can be estimated by assuring at least 1 milliliter per kilocalorie required or 30 milliliters per kilogram of body weight. Requirements will increase under conditions of great stress, fever, or infection.

To the best of my knowledge, vitamin and mineral requirements do not change significantly with age. Determination of vitamin and mineral requirements is difficult to perform in any way that is reliable, other than by surveying vast numbers of subjects and assessing vitamin nutriture and nutrient intake. These methods provide gross estimates at best and do not account for vast individual differences and the effects of chronic disease, interactions with medications, and subclinical deficiencies. Due to the physiologic ability to store the fat soluble vitamins, elderly people are at less risk of becoming deficient in these vitamins. It is possible for water soluble vitamin deficiencies to occur in the elderly due to inadequate intake of fruits and vegetables and a heavy reliance on calorie-rich, low nutrient density foods.

Mineral deficiencies probably occur as part of generalized malnutrition. Zinc, calcium, iron and magnesium deficiencies do occur in elderly persons, often associated with drug utilization, alcohol abuse and generally poor nutritional intake.

Conclusion

Aging people who have chronic illnesses, limited resources, live alone or are institutionalized may be at risk nutritionally. Loss of taste and smell sensitivities, poor dentition, and overuse of prescription and over-the-counter drugs also contribute to the development of nutritional deficiencies. All health care professionals should be aware of the impact of nutritional deficits on the health and well-being of elderly individuals and consider the importance of nutritionally sound diets when caring for them.

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Nutrition Throughout The Life Cycle: Discussion

Grossman, EM.

My first question is for Dr. Winick. You spoke of obesity in childhood being predictive of obesity in later life. Your talk seemed to have given pre-eminence to environmental factors in the causation of obesity. How about the role of genetic factors in obesity? To Dr. Chernoff, my question is this - Is it worse to be elderly and black than elderly and white? Do the black elderly have special, unique problems? I would also like to see Dr. Worthington-Roberts comment more fully on these racial differences in birth weights which she presented today.

Worthington-Roberts, B.

If I recall, the question relates to differences in susceptibility to the prenatal effects of alcohol, caffeine, and smoking. Black women as a group are much less likely to smoke than white women. There is good evidence for adverse effects of maternal alcohol intake on pregnancy outcome. I am not aware of any data suggesting racial differences in fetal handling of alcohol. In our part of the country, we certainly see a lot of native American offspring impacted upon by maternal alcohol intake. The issue of how vulnerable fetuses are to maternal caffeine use is still very debatable. A recent publication suggests that late first and second trimester spontaneous abortion is more common in women who use in excess of 150 mg. of caffeine daily.

Grossman, EM.

Dr. Winick, would you please address the problem of reconciliation of the obesity observations that you made with the genetic theories of obesity?

Winick, M.

Obesity is clearly a problem that has both genetic and environmental components, and to the extent that the genetic component is active, I suppose there is little that can be done to change it. Now, what evidence do we have that obesity has genetic components? The best evidence comes from studies on twins. Twins who are raised in an identical environment - be that environment either of obese parents or lean parents - the children will tend to have the same body build, either lean or obese if they are identical twins. With fraternal twins, the variance seems to be greater. If they are siblings and are raised in two different kinds of environment, then we begin to see the environment overwhelming the effects of genetics. To the extent that the environment is involved, that's really where childhood obesity becomes a serious problem.

When you say an individual has a genetic tendency to obesity, frankly you are not saying anything at all. You can still alter the expression of that genetic tendency by altering the environment.

Grossman, EM.

The last of my questions is for Dr. Chernoff, and has to do with whether there are special nutritional problems of the black elderly in contrast with the white elderly?

Chernoff, R.

I don't know that anyone has ever worked this out. One of the things that we don't see much in elderly blacks is osteoporosis, but we tend to see more hypertension and stroke. There are ongoing studies at Durham, which are looking at differences in minority aging.

Margen, S.

I would like to address a question to both Micky and Bonnie. This has to do with the relationship of food intake to maternal weight gain and the outcome of pregnancy in terms of birth weight. How much do you think we really can manipulate many of the pregnancies in terms of improving the birth outcome, by altering the nutrition? Micky, as I understood it, you said that an obese child at the age of 3 years has a 70% chance of being obese as an adult. Did you mean a 70% chance or a 70% increased risk?

Winick, M.

Seventy percent chance.

Worthington-Roberts, B.

Regarding the issue of weight gain as related to diet, there is no doubt in my mind that diet impacts upon maternal weight gain. There was in the last year's literature, a nice discussion of change in body composition in women in a developing part of the world where measures of fat pad change and weight change were recorded during the course of an entire year. In that particular part of the world, there are wet and dry seasons with two different states in terms of caloric availability. There was a distinct difference in fat deposition in these women as related to calorie availability.

Grossman, EM.

This is anecdotal but interesting. I was just reminded that my generation in medical school grew up with what was a simple advice - the baby weighed so much, the placenta weighed so much, and the amniotic fluid weighed so much - and the mother shouldn't gain any more weight than the sum total of those three weights. Of course, what everybody forgot at that time was that Mother Nature was busy laying fat storage for lactation. In my days as a student, if a woman gained more than 20 pounds during pregnancy, her obstetrician screamed at her.

Worthington-Roberts, B.

I wish to cite an example in Motherwell, Scotland which all of us in maternal and child nutrition know well about. For years, a physician there put women on a calorically restricted, high protein low carbohydrate diet during pregnancy. The babies that resulted from those pregnancies had birth weights 400 gm lower than babies in comparable communities without maternal caloric restriction in pregnancy.

Winick, M.

Evidence is overwhelming that caloric intake impacts on maternal weight gain during pregnancy and on the outcome. Even at the turn of the century, obstetricians knew very clearly that if they wanted to produce small babies, they had to reduce the amount of calories fed to the mothers.

Janet King has shown quite clearly that what is needed during pregnancy is 85,000 calories more on average for the extra caloric requirement. For tissue deposition, we are talking of 5,000 - 7,500 calories necessary to deposit all the lean tissue in the baby, placenta and uterus. So, the caloric requirement for growth of the entire conceptus and the increase of the mother is trivial. That leaves us with two components. One is the increased metabolic activity of both the mother and fetus during pregnancy. Early in pregnancy, maternal metabolic activity goes up. Later in pregnancy, fetal metabolism is quite rapid - more so than the maternal metabolism. These account for about half of those extra calories. The third component has to do with deposition of extra maternal body fat.

Now the answer to your second question. How do I define obesity at age three. The definition would fall into two possible categories. The easiest definition is 120% or greater of ideal body weight for that age. Such a child has 60 - 70 per cent chance of becoming an obese adult. If you look at the data from Jules Hirsch, children who are significantly obese at age 3, usually have a hyperplastic obesity - some of them have more fat cells than a normal adult.

Margen, S.

I tend to agree with both of you. I raised the question because of recent work from Whitehead and his group at the Dunn who feel that the total intake required is probably around 20,000 calories instead of 80,000 - 90,000. I really wanted the issue emphasized because I feel very much as you both do that nutrition during pregnancy is a very important factor and I don't want to see the importance minimized as a result of some recently published work.

One last question, would you comment again on this obesity issue in the light of some work published some time ago by Stan Garn, showing that the correlation between childhood obesity and adult obesity was virtually nil?

Winick, M.

Let me first comment on the Whitehead study in Gambia. Those women were involved in work pattern activities completely different from what obtains in this country. How these work pattern/activities impact on caloric intake and birth weight are not really clear.

Now Stan Garn's data are very strong, but they relate to children below 3 years of age when there is very poor correlation between childhood obesity and obesity in adult life.

Worthington-Roberts, B.

I am amazed at the weight gain you have recommended for pregnant teens, Dr. Winick. In our clinic, about 25 per cent of the pregnant teens start pregnancy obese and with a gynecologic age of about 3 - 4 years. What would you recommend as an appropriate weight gain in this situation?

Winick, M.

I hope that I didn't give the impression that a teenage girl should gain 60 lbs. What I was saying was that if in fact one wanted to use those statistics in underweight teenagers to predict the best outcome in terms of birth weight, the teenage pregnant would have to gain 60 lbs. That is something that none of us would want to see her gain. By definition, all teenagers who are underweight before they become pregnant, and who have to grow at the same time, will go through "malnourished pregnancy", and that is why they have smaller babies. I wasn't advocating correcting this by letting her gain 60 or 80 lbs.

The next question on how much weight obese individuals should gain during pregnancy - the data there are weak at best. We know that if an obese woman looses weight during pregnancy, she will have a small baby. I am talking of actual weight loss to the extent that she ends up weighing significantly less than she did before pregnancy. Now, where do I make that statement from? I guess the best and most dramatic study is a study of women who have had intestinal by-pass and who are therefore unable to increase their caloric intake no matter what you do to them. Such women deliver babies that are not only small, but they have smaller babies than non-obese women, with average birth weight dropping to less than 3,000 gm. The standard recommended weight gain for obese women during pregnancy is 15 lbs. This has been confirmed by Rosso and his group.

Natta, C.

My first comment has to do with taking fasting blood glucose of 140 mg as normal in a woman who is in her 70s or 80s. The problem that we have seen with the black patients in our Harlem hospital is this. If there is a family history, invariably when you do a glucose tolerance test, the one hour point is over 200 mg. These patients usually have recurrent urinary tract infection, and we find that we can control that by dietary means or oral agents.

The second point is with reference to your folic acid data. Now among the black patients in Harlem hospital, we have seen a lot of borderline hemoglobin and hematocrit, as well as low mean cell volume and when you work them up, they have no underlying illness except probably x-thalassemia G. We think they are relatively folic acid deficient because they respond to folic acid. We have not done estimation of red cell or serum folate.

Chernoff, R.

There are a lot of elderly people who have decreased glucose tolerance, with fasting blood sugar of about 130 - 140 mg, and who are not diabetic. They don't necessarily have to be treated.

Regarding the comment about folic acid, you are dealing with a group who probably have higher needs for folic acid because of a medical problem. There is something called the "anemia of senescence" where hematocrit and hemoglobin levels tend to decrease slightly over time simply because of old age, and the inability of the bone marrow to function as well as it did in earlier years.

Natta, C.

The question of folate is also complicated by the fact that a 75 year old person who has atrophic gastritis does not absorb vitamin B12 very well. B12 would affect the folic acid status. In the black population, there is also the question of G6PD deficiency, which over a period of time will produce changes in the folate level.

Winick, M.

Let me just make a comment if I may about folate in the elderly. One of our graduate students has found that in the well, older individual with no evidence of any disease, there is a defect in the ability of red cells to take up folate. Thus, serum folate levels may in fact be higher than in the young population, and vice versa for red cell folate levels. If you give a folic acid challenge to such an individual, you raise the serum folate levels immediately, but the increase in red cell folate levels takes a good deal longer, and does not reach the level that one would expect in a younger population. This worker has coined the use of a "folate index" which is the ratio of serum to red cell folate. Folate index is much higher in the well elderly than in the younger population.

I certainly agree with you that B12 status of these individuals would be critical in terms of their folic acid status. I am also intrigued by the possibility that people who are heterozygotes for a particular hemoglobinopathy, may in old age act differently in terms of the metabolism of nutrients.

Grossman, EM.

I think the point on vitamin A mentioned by Dr. Chernoff deserves emphasis. The elderly are very given to taking vitamin supplements. Some studies have shown that as high as 65% of them in some areas of the country take vitamin supplements.

Dr. Russell (USDA, Boston) has shown that as we get older, we have an increased uptake of vitamin A, and a diminished ability of the liver to process it, so that one ends up with increased retinyl ester stores in the liver, which may approach toxic levels in people who have been taking vitamin supplement (5,000 units of A) every day for 4 - 5 years.

Chernoff, R.

I mentioned that I have reviewed the AARP over-the-counter handbook, and the amount of vitamin A in some of those vitamin supplements may be up to 20,000 - 25,000 IU range.

Anonymous.

My question is to Dr. Winick. In the individual with infantile marasmus, does a change in diet after the period of active brain development correct retardation?

Winick, M.

Clearly this has not been done in people, but the answer in animals is very clearly no. Once the active period of brain cell division has passed nothing you can do will increase the number of cells. However, there are things that we can do given the fact the infant is going to have fewer cells. For an infant who has had marasmus, we can insure that the infant's development is not at least impaired to any extent as great as the child who is in a poor environment by providing such a child with a good environment. This raises some important clinical questions that people in the neonatalogy business never have had to deal with before. How aggressive should one be, for example, in hypoalimenting a very small malnourished premature infant in terms of preserving brain growth from a practical standpoint? Really, it depends on what kind of an environment that that premature infant is going to grow up in. That is not the way decisions are made in premature nurseries. So it is a very complex problem in terms of what kind of management one would institute. But to answer your question, once cell division stops there is no way that we know of to turn it back on.

Worthington-Roberts, B.

Just a quick comment. It will be very interesting to see the ultimate result of studies that are going on right now to follow babies of very low birth weight who have survived and are now in school. The data that we have from own our environment suggest very strongly that a good 50% of these very tiny survivors have problems that do not really surface until exposed to the academic environment or to even the social environment of a preschool setting.

Kinnard, M.

Dr Winick, it has been my opinion that there has been an overemphasis on cranial capacity, decreased number of brain cells and IQ. Is it not possible that decrease in the size of cranial capacity could be something that is genetic and has nothing to do with intelligence?

Winick, M.

I don't think that there is any question that the number of cells in an organ is primarily under genetic control. So I don't think that one can the try to correlate cranial size with intelligence and come out with a meaningful kind of correlation. On the other hand, if you take an organ and you retard its growth and then you go ahead and ask the question, can you correlate that stunting with some function of the organ, it depends on what function you are looking at. Clearly if you stunt the liver, you are not going to be able to find any functional impairment because 80% of the liver will do just as well as 100% of the liver. When we get to the brain it becomes a complex question. We may not see any functional impact if the animal with fewer brain cells grows in the proper environment. I will also point out that when malnutrition occurs, different parts of the brain are affected differently depending upon the rate of cell division that happens to be going on in the area at that time. So, if I take a rat and malnourish it for the first 21 days of life, I have affected the cerebellum more than the cerebrum. What are the consequences of having one part of the brain affected more than another part? Again, we don't fully know. We can't demonstrate these changes as major changes if the animal is reared in a good environment.

Kinnard, M.

My last question is to Dr. Chernoff. Is there sexual dichotomy in the aging process?

Chernoff, R.

I am not aware that this has been demonstrated. I don't for example think we know why women tend to live longer than men. I really don't think it has anything to do with nutrition.

THIRD SCIENTIFIC SESSION

Nutrition And Chronic Diseases

CHAIRPERSON:

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Nutrition As A Risk Factor in Hypertension and Cardiovascular Disease

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Despite the fact that great progress has been made in the prevention and treatment of cardiovascular disease, it remains one of our most serious health problems, affecting hundreds of thousands of people in this country every year. The consequences of essential hypertension and coronary artery disease are grave, indeed, affecting as they do, so many of the vital organ systems and, of course, life itself.

Few people would dispute the statement that, in greater or lesser degree, nutritional factors are intimately involved with the onset and course of hypertension and coronary disease. In the case of essential hypertension such nutritional parameters as obesity, sodium intake, and alcohol come to mind immediately but the roles of potassium and calcium in the diet are also given prominence by experts in the field. Similarly, obesity, diabetes, and the role of saturated fats in the diet occupy a very important place in any discussion of coronary heart disease. I'm sure other examples of the role of diet in the prevention and treatment of these conditions will occur to you as well.

But, in addition to sharing all of these risk factors with their fellow Americans, there are special problems affecting the black population and it is to these special problems that I will direct my attention today.

First, a brief look at some of the statistics. NIH reports that an estimated 6.8 million blacks nationwide have high blood pressure (1).

Blacks are said to be 5-7 times more likely to suffer from severe hypertension and 17 times more likely to have secondary complications from unchecked high blood pressure (1). Stroke, for instance, affects about 500,000 Americans a year, killing 155,000 of them. Blacks are over 1-1 1/2 times more likely than whites to develop strokes. And, among blacks, the narrowing of the arteries that is found inside the skull is more common in women, whereas, in whites of both sexes, carotid artery disease (extracranial) is far more common. Another interesting difference that has recently been reported is that, in blacks, the risk factor was high blood pressure. White stroke victims, on the other hand, often have coronary artery disease and elevated cholesterol (2).

Right here at Meharry, we have had the benefit of an ongoing study by John Thomas, M.D. Dr. Thomas and co-workers have been following a group of 313 black male Meharry Medical College physicians and a matched cohort of 470 white male graduates from the Johns Hopkins School of Medicine. Now, more than 20 years after medical school, the rate of heart attack among these black physicians is 10 times greater and the incidence of hypertension is three times higher. Dr. Thomas is quoted as saying that "this study shows that being of a high socioeconomic group does not protect black physicians to the extent that it appears to do for the general population"(3). The increased incidence of hypertension, alone, did not explain the prevalence of heart disease, although smoking and weight gain did appear to be factors of some significance. However, it should be noted that 47% of the black M.D.'s developed hypertension versus only 14.3% for the whites. The study is continuing. In a survey of "Elevated Blood Pressure Among Harlem Residents," Brown et al (4) reported finding 40% of those screened had blood pressures above the normal range for their age, which is a rate twice that of the reported incidence in the general population of the U.S.

As far as coronary heart disease is concerned, the picture is more complicated. Coronary heart disease is relatively rare in black Africa and is less extensive among black populations in the Carribean than in the U.S.(5) where coronary heart disease is the leading cause of death among blacks(6). In South Africa, on the other hand, essential hypertension is a severe public health problem reaching 25% among urban blacks(7). These statistics cannot be interpreted in terms of genetic susceptibility, alone.

There may be one bright note in all of this otherwise gloomy news. According to Persky et al(8), the non-white/white ratios for mortality from complications of hypertension decreased between 1968 and 1978. Prevalence data show a corresponding recent decrease in black minus white mean blood pressure; some of the decrease appears to be due to a greater improvement in hypertension control for blacks than for whites.

Having seen that essential hypertension, HCVD, and coronary heart disease, in general, are diseases of great concern to blacks, it is opportune at this juncture, to explore the possible contributions of dietary practices to these problems. There are two lines of evidence which may help us to understand the special vulnerability of blacks to hypertension and coronary heart disease. The first is one in which dietary surveys are conducted and an attempt is made to detect black-white differences in the use of sodium, potassium, and other substances, such as calcium, which have relevance to the disease process. A corollary here might be to stage an intervention trial modifying the individual's habitual intake and noting any effects on B.P.

The second type of study looks for physiologic differences in the handling of various mineral loads; such studies are usually driven by the hypothesis that a genetic factor is responsible for the special vulnerability of some blacks in Western society to certain forms of cardiovascular disease. Again, the implications for diet are obvious.

In 1975, Desor et al found that young black children preferred stronger concentrations of sodium chloride than whites(9). This preference tended to disappear in the adults. The Bogalusa Heart Study(10) reported that black girls had a significantly greater mean sodium intake than black boys or white boys and girls. Harlan et al (11), in reviewing NHANES-I found that older blacks had the highest rates of high blood pressure, as expected. However, there was no association with dietary sodium or salt use. Frisancho et al(12), working from the same data base, noted that, while hypertension did not appear to be related to an absolutely greater dietary sodium intake, it was related to a relatively low intake of potassium.

In 1977, Luft et al(13) studied the responses of a group of normotensive black and white subjects in their response to a salt load. They found "racial differences in renal sodium excretion reflected by decreased urinary sodium and a relative suppression of plasma renin activity following a salt load in blacks". Five years later, Weinberger et al(14) found that "blood pressure increased at lower levels of sodium intake and to a greater magnitude in blacks than in whites." Both these authors and others(15) noted that potassium administration could affect sodium balance favorably, more so in blacks than whites.

In a recently published clinical trial of potassium chloride in the treatment of mild hypertension, Svetkey(16) et al concluded that "blacks may be particularly sensitive to blood pressure - lowering effects of potassium chloride".

Another dietary mineral which has attracted great attention recently for its possible role in hypertension, is calcium. McGarron(17), as we know, has been a vigorous champion for assigning a primary role to calcium in hypertension. Several groups of investigators have put his hypothesis to the test by administering calcium supplements to black and white subjects. In normotensives, Lyle et al(18) reported no significant difference in response between the two groups of subjects. However, 1500 mgs. of calcium a day produced a lower mean arterial pressure during a 12-week trial. These differences were small, 2-3 mm. Hg, and were not correlated with any changes in laboratory measurements of serum calcium, phosphorus, or parathyroid hormone.

Zemel and co-workers at Wayne State University in Detroit have shown(19) that increasing dietary calcium attenuates salt-induced blood pressure elevation in salt-sensitive blacks. Studies of the mechanisms appear to involve a "calcium-induced natriuresis and subsequent inhibition of sodium-induced volume expansion." A high dietary sodium intake appears to cause PTH-mediated increase in intracellular calcium which is prevented by supplementary dietary calcium. From my review of their data, it would appear that one gram of calcium can neutralize the blood pressure-raising effect of 4 grams of dietary sodium.

In more recent work, appearing thus far only in abstract form(20), Dr. Zemel and colleagues have demonstrated a trend toward decreased serum total calcium with age in the black population, together with a rising PTH level. According to the authors, this age-associated increase in PTH in black males may explain, in part, their greater incidence of age-related hypertension, as elevated PTH levels are frequently associated with blood pressure elevations.

In the past few years, evidence has accumulated that yet another mineral plays a role in essential hypertension: magnesium. A precise role has yet to be defined since one can find both positive and negative results in the literature so far as clinical intervention studies are concerned. The most reasonable conclusion seems to be that of Wester and Dyckner who write, in a recent paper appearing in the American Journal of Clinical Nutrition(21):

"although there is no evidence that peroral Mg supplementation alone, lowers BP in untreated...patients, we have demonstrated that addition of Mg to the antihypertensive regimen (thiazide plus K) of...patients further lowered their BP".

To sum up the work that I have discussed so far, some blacks would appear to have a genetic sensitivity to a high sodium intake. To the extent that they partake of the high salt intake so common in Western diets, they will be more susceptible to the development of essential hypertension and its pathological consequences: stroke, heart disease, and renal failure. But it is these very individuals who are amenable to modification of the hypertension process because it appears to be favorably influenced by the reduction of the sodium load, by supplementation with potassium, or the addition of calcium, and, possibly, magnesium. Surely, there are lessons here for every therapeutic dietitian. To lower the sodium load, one needs to merely avoid those foods to which an excess of salt has been added before it is presented to the consumer. To raise potassium intake, fruit, fruit juice, and vegetable consumption need to be encouraged. Calcium intake always presents a problem where dairy products must be limited because of lactose intolerance. The use of lactose enzymes added to whole milk; yogurt consumption; limited amounts of cheese, milk, etc; and the use of broccoli and collards can be encouraged to raise calcium intake. In appropriate situations, it could be advisable to prescribe a calcium supplement and if it is efficacious, it would certainly be preferable to long-term antihypertensive medication. Foods rich in magnesium may also have a role to play.

As for the other dietary factors, I will merely make passing reference to an assortment of nutritional elements for which there is at least some evidence that they have a role in hypertension. I refer to such factors as obesity, excessive alcohol intake and saturated fats. Together with the well-recognized dietary factors that are associated with coronary heart disease (i.e. - saturated fats, cholesterol) they should be recognized as playing precisely the same part in the pathogenesis of black cardiovascular disease as they do in whites and, as such, present us with an opportunity to prevent or modify the course of these diseases.

Of interest, therapeutically, is the paper by Connett and Stamler(22) where some data from the MRFIT program are examined and the conclusion is reached that they indicated "ability to induce extensive changes in life-styles (eating and smoking habits) of both black and white American males of varying educational and socio-economic backgrounds and also to achieve long-term effective adherence to programs for the treatment and control of hypertension."

No discussion of the factors leading to the increased prevalence of hypertensive cardiovascular disease among blacks in the United States would be complete without some reference to their socio-economic status. The very process of selection of a diet from a wide array of possible choices is strongly influenced by one's income and one's ethnic origins. In addition, the interaction of dietary intake with stress factors has been examined by Duh et al in their paper on "An Ecological View of Hypertension in Blacks"(23) and I would commend their views to you for careful study if you would hope to gain a broader understanding of the problem of nutritional influences on the cardiovascular problems of blacks. These authors point out that the difference in prevalence of hypertension between black Americans and black Africans - and between those living in urban and rural areas - cannot be explained by genetic factors, alone. A more complete appraisal of all known risk factors must include the contribution of stress to the etiology and maintenance of the hypertensive condition. So, all in all, I take a rather optimistic view of what can be done to increase our understanding of - and improve the outlook for - the management of cardiovascular problems in black Americans. It is possible to modify many of the risk factors. A multifaceted approach will be necessary, including education, dietary modification, and life style changes but the problems are not insoluble.

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Obesity In Blacks

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This talk will be in two parts. The first section will review what is known about obesity in blacks, and whether it is different in any respect from obesity in caucasians? Second I will share with you some of the new studies which probe energy balance in obese and normal subjects.

In the USA obesity in adults varies according to sex and race. Thus obesity is most prevalent in black women, than white women and white men, and least in black men. This may partly be explained by socioeconomic stratification, since persons in a high income bracket have a 7% incidence of obesity, while persons in a low income bracket have a >30% incidence of obesity. Obese individuals tend to move down the socioeconomic ladder(1). In the middle of the 19th century settlers from Europe were frequently obese for it was a symbol of their economic success. In subsequent generations affluence has been marked by increasing slimness. Thus obesity in persons of higher socioeconomic status decreases as the number of family generations in the US increases. Discrimination against obese individuals is not too subtle and the discrimination is more against women than men.

Thus in a study of admission to eastern seaboard universities, the likelihood of being accepted was 90% for normal weight males and 85% for obese males, 92% for normal weight females and 58% for obese females. There is no relationship between weight and intelligence and thus the lowered acceptance rate, especially of obese women, reflects a prejudice against overweight persons. This prejudice is very general and undoubtedly explains in large measure why obese individuals, especially women, move down the socioeconomic scale.

Studies by Gortmaker, Dietz, et al, reviewing data from the national nutrition surveys (HANES I&II) show that the percentage of "super obese" (>95% weight for height) children between 6-11 and 12-17 years has increased during the past 20 years(2) [See Figure 1].

While obesity is more common in black than white children, when the children are compared according to economic status the percentage of poor white children who are obese is greater than the percentage of poor black children. However numerically black obese children predominate because more black children are poor.

Morbidity and mortality in relation to weight are expressed by a J shaped curve. They are increased in individuals who are less than 10% of their average age weight and are increased again as the individual is more than 25% above his average weight. Increased morbidity and mortality due to excess weight is high in young individuals and has much less prognostic value in older individuals. For example, a twenty-year-old man who is 100 pounds overweight has a 12-fold greater likelihood of dying, while a seventy year old man has only twice the likelihood of dying, compared to their normal weight counterparts. The recent NIH publication entitled "Health Implications of Obesity" has provided figures for the excess prevalence of certain obesity associated disorders (3). Hypertension is several fold more common in obese individuals, especially in younger age groups where it is six times more common than in normal weight persons. Hypercholesterolemia, non-insulin dependent diabetes mellitus, and cancer of the colon, bile ducts, and in women, of the breast and uterus are all more prevalent in the obese. A study of Meharry medical students, who were normotensive and only 7% obese at the initial evaluation found that 22 years later 18% were obese and 44% of these black physicians had developed hypertension(4). There was a strong association between developing obesity and developing hypertension. Studies of black children have shown the same association between developing obesity and developing hypertension(5). In the very large cohort of black and caucasian children evaluated for lipoprotein patterns by Berensons group(6), triglycerides increased with increasing obesity but total cholesterol was unchanged (Figure 2).

However as the weight increased the cholesterol was greater in the LDL fraction and less in the HDL fraction, so that LDL/HDL cholesterol ratio increased and this, of course, is a predictor for greater coronary artery disease.

It is interesting that there may be certain types of genetic obesity where excess weight is not linked to excess morbidity and mortality. Working in South Africa, Walker has described the adolescent onset of obesity in girls of certain African tribes while boys from the same tribes stay very lean(7-9). In hospital workers from these same tribes, women working as nurses had an incidence of obesity close to 90% while men from the same tribes working as orderlies were rarely ever overweight. This female obesity was not associated with serious prognostic factors such as hypercholesterolemia, hyperuricemia, nor with hypertension or increased risk of certain cancers. While Walker did not describe the distribution of adiposity in these African women it is tempting to speculate that the obesity may have been more peripheral than central, a distribution known to have less adverse health implications, as pointed out by Kisselbach(10). Why would adiposity in the femoral and gluteal areas, a type of exaggerated female secondary sexual distribution of fat, be less associated with obesity related diseases than adiposity around the waist and upper trunk? The current hypothesis is that perivisceral fat depots increase the free fatty acid concentration of portal blood. It is known that as the portal vein free fatty acid concentration rises hepatic clearance of insulin decreases. The liver normally clears about 80% of insulin secreted by the pancreas. In obese individuals the increased insulin escaping into the systemic circulation may act as a potent stimulus to lipogenesis and cause down regulation of insulin receptors and hence greater insulin resistance, leading to glucose intolerance and diabetes. Hyperinsulinemia may also play a role in regulating sodium retention by the kidneys and hence contributing to hypertension. While this sequence of events is only a theoretical explanation for the adverse implication of central obesity, it offers a testable hypothesis.

Turning now to the second part of this talk - what are the metabolic derangements leading to obesity? Obviously it is an *a priori* truth that obese persons are in positive energy balance. Either they take in too many calories or they expend too few. Recent data suggest that the type of calorie and possibly the time of day the calorie is eaten can influence the net calories stored. Calories derived from fat involve very little energy for their absorption, transport and storage. According to calculations made by Flatt, only 3 percent of the initial food calories are expended in processing when the food is triglyceride(11). If, on the other hand, the calories come from complex carbohydrate, which involves hydrolysis and then storage as glycogen or lipid, 15% of the initial calories are consumed in food processing. If calories are derived chiefly from protein sources and require gluconeogenesis for conversion to an energy source, then 25% of the initial calories are lost in energy processing. Thus the source of dietary calories matters and individuals consuming a large percentage of their calories as fat are more likely to be in positive net energy balance. It is worth noting that in the past 50 years the average daily calorie intake has decreased approximately by 1000 calories, reflecting our more sedentary lifestyles compared with our grandparents. However the percentage of calories consumed as fat has relatively increased from about 30% to 45% . This change could in part explain the current epidemic of obesity. To date there are no reliable and sensitive techniques for measuring food intake in free living individuals, but there are a number of studies done in inpatient research settings which show that many obese persons do not consume significantly more calories than their lean counterparts, although they appear to be less sensitive to satiety signals.

Measuring total energy expenditure can now be done with considerable accuracy. Direct calorimeters which measure total heat exchange are necessarily very small in dimension to attain reasonable sensitivity and therefore are not well suited to evaluating persons under normal living conditions. Indirect calorimetry, which calculates energy expenditure from oxygen consumption and carbon dioxide excretion, can be used in relatively free living conditions for either short periods using the Douglas bag or for longer periods in a large respiratory chamber as exists in Lausanne, Switzerland and Phoenix, Arizona. Study subjects can live in these respiratory chambers for 24 hours to several days and their activity is recorded by a body oscillometer and radar cameras. These two centers have published data on large numbers of normal and obese subjects. The studies in Phoenix focus particularly on Pima Indians who are genetically, under modern living conditions, very prone to obesity (75% adults) and non insulin dependent diabetes (50% adults). A third method for measuring expenditure is the doubly labeled isotopic water technique. This method can be used in totally free living individuals measuring their total energy expenditure over 7 to 10 days. Because it uses stable isotopes in minute quantities, the test is safe and has potential for evaluating a broad range of subjects, including children and pregnant women. Results using the isotopic technique agree with direct and indirect calorimetric measurements to within 5%. Thus far only a few studies have been done with this technique but it holds great future promise.

What have we learned to date about energy expenditure in lean and obese subjects? Studies published by Bogardus et al (12, 13) from Phoenix, show that resting metabolic rate (RMR) and 24 hr. energy expenditure (24 hr. E.E.) in the respiratory chamber correlate very strongly with fat free mass, age and sex, the fat free mass being the most important determinant (Figure 3).

These three factors account for >80% of the intersubject variance. Familial membership accounts for an additional 11% of the variance, showing that parameters of energy expenditure are part of a familial trait. Initially these investigators found no simple correlation between obesity and lower resting metabolic rates, implying that there are fat families who eat little and expend little and there are obese families who eat a lot and expend a lot. After repeating these same measurements on an annual basis for 4 years, it is clear however that individuals with a low energy expenditure (RMR or 24 hr. EE) are at far greater risk for gaining a significant amount of weight than individuals with average or above average energy expenditures(14). As their weights increase these obese individuals develop a greater daily energy expenditure per kilogram of fat free mass. An increase in fat free mass is expected as weight increases but the increase of expenditure per kilogram of fat free mass is less easily explained and may reflect increased gluconeogenesis, increased rates of protein turnover, substrate recycling and sympathetic nervous system activity. Thus weight gain may induce compensatory mechanisms which eventually match any increased energy intake and allow the subject to restabilize at a new weight. Chamber studies have shown great intersubject variation in spontaneous activity or "fidgeting". Such activity can account for as little as 100 cals/day, to over 800 cals/day.

While reduced energy expenditure appears to be a significant risk factor for body weight gain and may contribute to the aggregation of obesity in families, it can be calculated that such intersubject differences account for only about 40% of the experienced weight increase, and thus other important factors, such as calorie intake and differences in physical activity in free living persons must also have an important role in regulation of body weight.

A recent study in infants(15) using the doubly labeled water method, has shown that infants born to overweight women have a total energy expenditure, 20% lower than infants of normal weight mothers at 3 months of age. While at 3 months both groups of infants were the same weight for length, by 12 months 50% of the underexpending infants were above the 90% weight for length (Figure 4). This important study confirms the implications of the Ravussin data (12, 14) that obese subjects have a metabolic deficit in energy expenditure which precedes weight gain.

Since weight reduction is extremely difficult to achieve and sustain in obese subjects, short of invasive surgery, probably the thrust of obesity care should go into obesity prevention and be a public health challenge to stimulate physical activity and reduce energy intake, particularly fat intake in the individuals who are genetically prone(16). Family studies have shown that 2 obese parents mean 80% of children are at risk, no obese parent 7% of children are at risk and one obese parent 40-50% of children are at risk.

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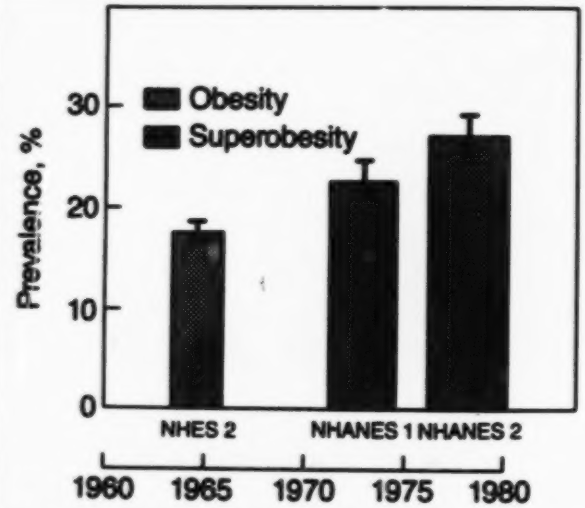


Fig 1.—Estimated trends in obesity and superobesity in children 6 to 11 years old in United States. Bars indicate approximate 95% confidence intervals.

Figure 1

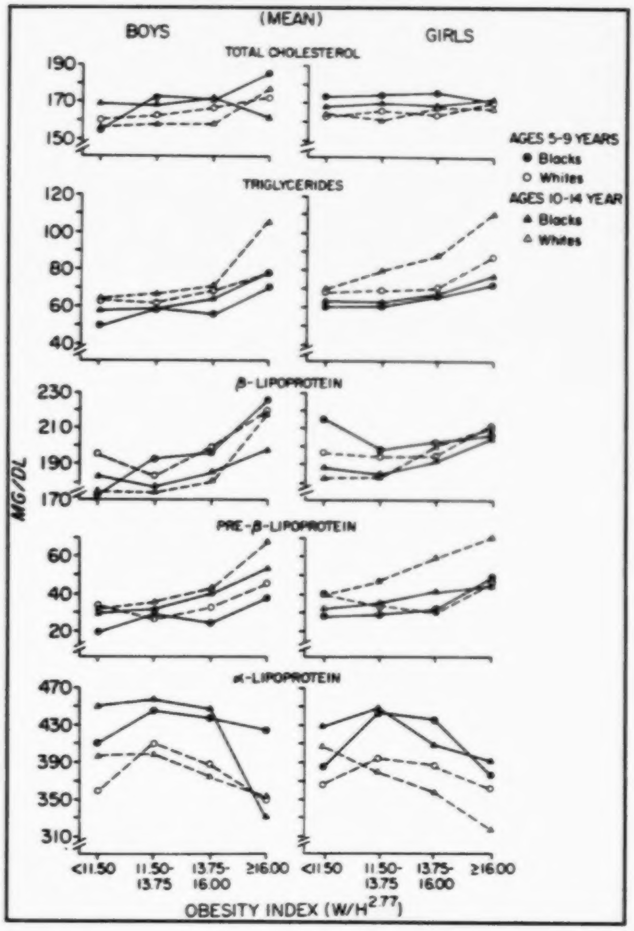
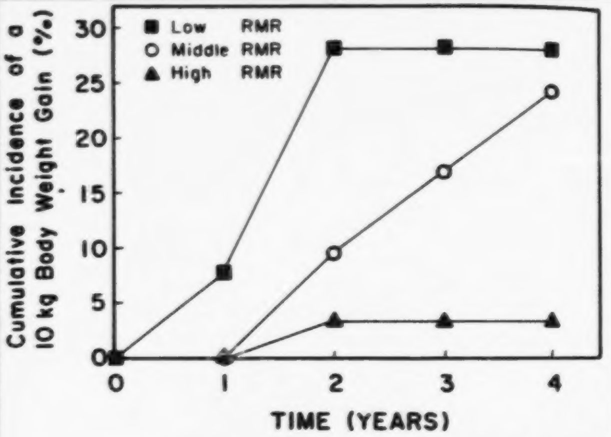


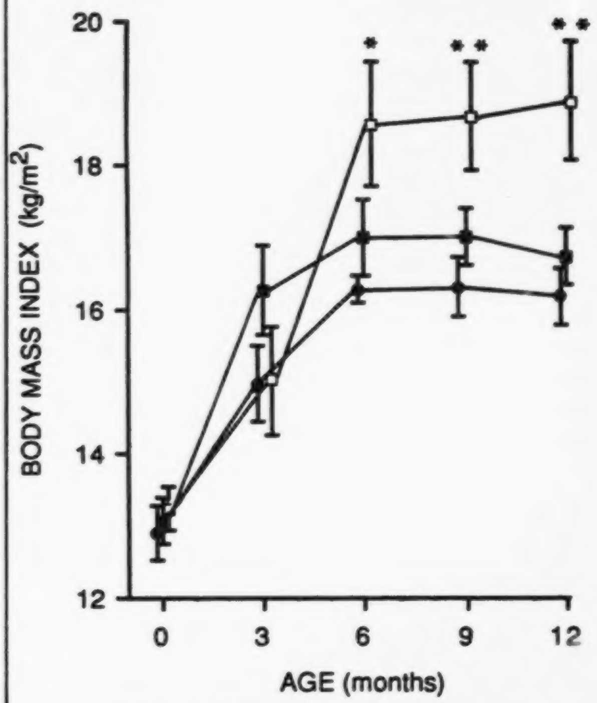
Figure 2



Cumulative Incidence of a 10-Kg Gain in Body Weight in Three Groups of 42 Subjects.

The groups were defined according to tertiles of metabolic rate at rest (RMR), adjusted for fat-free body mass, fat mass, age, and sex. A low RMR was defined as ≤1705 kcal per day, a middle RMR as 1706 to 1797 kcal per day, and a high RMR as ≥1798 kcal per day. One hundred twenty-six subjects were followed for at least one year, 89 for at least two years, 49 for at least three years, and 13 for four years.

Figure 3



. Body-Mass Index from 0.1 to 12 Months of Age in Infants Born to Lean (●——●) and Overweight Mothers.

Infants born to overweight mothers are divided into those who became overweight during the study (□——□) and those who did not (■——■). Values are means ±SE; n = 6 per subgroup. There was no significant difference between normal infants born to lean mothers and normal infants born to overweight mothers. Infants who became overweight had significantly higher values than all normal infants combined at 6, 9, and 12 months of age. The single asterisk indicates $P<0.05$, and the double asterisk $P<0.01$.

Figure 4

Nutrition and Diabetes Mellitus

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Diabetes mellitus is a serious chronic disease in the United States. More then 150,000 deaths each year are due to diabetes and its complications (1). Quality of life and the economic costs of the disease are major concerns. Diabetes leads to severe health complications - blindness, kidney disease, amputations, coronary artery disease, peripheral vascular disease, and stroke. Improved treatment and management can lead to better control and lifestyle.

The economic impact of diabetes to the nation remains at $14 billion annually (1). This conservative figure accounts for medical costs, disability and absences from work, and impairment of normal daily activities.

Noninsulin-Dependent Diabetes Mellitus (NDDM)

Eighty to ninety percent of the individuals with diabetes in the United States have noninsulin-dependent diabetes (NIDDM). These persons are usually older than 40 years at diagnosis and 70 to 90 percent are obese or have a history of obesity at the time of diagnosis. The prevalence of NIDDM in the United States is over 5 million persons or about 2.5 percent of the population. There are believed to be an equal number of undiagnosed cases. It is now present in 20 percent of individuals over age 65. The prevalence rate increases with age and degree of obesity. Although the majority of patients with diabetes are Caucasian, it is reported tht 15 percent of adults with diabetes are black and 6 percent of Hispanic origin. Half of the Pima Indians over age 35 have diabetes (2).

NIDDm and Imparied Glucose Tolerance (IGT) are erroneously believed to be the "mild" form of this disease. The public, as well as health professionals, often believe they are not associated with the long-term complications and are us only if insulin is required. Unfortunately, this is simply not the case. The number of complications that afflict persons with NIDDM is extraordinarily long. There is strong, but as yet not conclusive, evidence that good metabolic control will prevent the development of long-term diabetic complications. Vigorous attempts to normalize blood glucose should be part of the management of persons with NIDDM and IGT before the major complications of diabetes develop (3).

Family studies have clearly indicated that insulin-dependent (IDDM) and NIDDM are distinct genetic disorders. Among monozygotic twin pairs with NIDDM concordance approaches 100% suggesting that genetic factors play an important factor in the pathogenesis.

Theories on the etiology of NIDDM relate the development of the hyperglycemia to four possible causes:

1. Impaired insulin secretion. It is proposed that in NIDDM the defect in insulin secretion is partially compensated for by

the resetting of the plasma glucose concentration to a higher steady- state level. There is no first-phase insulin release in response to food and the second-phase insulin release occurs but is blunted.

2.Impaired insulin action (insulin resistance).
Insulin resistance results in defects in the process of insulin binding to cell membrane receptor sites as well as the generation of post receptor defects.

3. Excess endogenous glucose production (increasedhepatic glucose output). Excess hepatic outputof glucose is primarily responsible for the elevated fasting levels of plasma glucose.

4. Excess exogenous glucose (caloric intake). It is estimated that control of obesity through diet, weight loss and exercise could prevent up to 50% of the cases of NIDDM or 293,000 cases annually(4).

Insulin-Dependent Diabetes Mellitus (IDDM)
Insulin-dependent diabetes mellitus occurs in approximately 10% of the individuals with diabetes (2). Current research suggests that the etiology of this disease is multifactorial. In genetically susceptible humans, an environmental stimulus may provoke an autoimmune response resulting in the destruction of islet cells.

Management of Diabetes Melliuts

Management of both types of diabetes involves several factors:

1. Education - multidisciplinary approach including the physician, dietitian, nurse, family counselor, etc.
2. Nutrition
3. Exercise
4. blood glucose monitoring
5. Oral hypoglycemic agents (NIDDM)
6. Insulin (IDDM and possibly NIDDM)
7. Treatment of associated disorders, e.g. hypertension, hyperlipidemia, etc.

Nutrition

The role of nutrition in diabetes management has recently been reexamined. Updated nutritional recommendations and principles of nutrition management have been published by the American Diabetes Association (5). Education begins at diagnosis with initial or survival level education. Once the adjustment process has begun, the individual is ready for more in-depth education. Continuing education is essential if the individual is to incorporate diabetes into his/her lifestyle and to manage his/her diabetes successfully (6).

1986 Nutrition Recommendations

Calories

Calories should be prescribed to achieve and maintain a desirable body weight. Although the recommendations use the phrase "desirable body weight", the patient education materials use the phrase "reasonable body weight". Reasonable weight is used because the weight goal of an individual may not be desirable or ideal. An individual will have both short-term and long-term goals for a reasonable weight. For example, Mr. Jones weighs 250 pounds. His desirable body weight is 175, but a reasonable weight goal may be 230 pounds. After that is achieved, the reasonable weight goal may change to 210 pounds, and so on. Using "reasonable body weight" allows the nutrition counselor and patient to set achievable and realistic goals.

For the person with noninsulin-dependent diabetes mellitus (NIDDM), the cornerstone of therapy is a lifestyle centered around diet and exercise and supplemented, if needed, by insulin or oral hypoglycemic agents. Blood glucose levels usually return to normal as weight loss occurs in the obese person with diabetes. Weight loss also improves hypertension, hypertriglyceridemia, and hypercholesterolemia.

For persons with insulin-dependent diabetes (IDDM) who are usually lean at diagnosis, it is important that adequate calories be prescribed. This is of special concern for children and adolescents. The biggest mistake made in prescribing meal plans for this group is usually inadequate calorie levels. Calorie levels should be based on the nutrition history and usual food intake.

Carbohydrate

The amount of carbohydrate should be liberalized, ideally it can be up to 55-60 percent of the total calories. This recommendation does not state that the meal plan must contain 55 to 60 percent of total calories from carbohydrate, but instead that the percentage is to be individualized with the amount dependent on the individual's previous eating habits and its impact on blood glucose and lipid levels. The emphasis is on individualization - making changes that are important to achieve diabetes management goals. What changes are the individual willing and able to make in his/her usual eating habits?

The substitution of fiber containing food for food with highly refined carbohydrate, which is usually also low in fiber, is encouraged. Water-soluble fiber (found in legumes, oats, barley, and some fruits) appears to form a gel within the gastro-intestinal tract, thus slowing the absorption of glucose across the intestinal mucosa as well as lowering total cholesterol an LDL-cholesterol. Water-insoluble fiber (wheat and corn bran, whole grains, and some vegetables) increases bulk within the gastro-intestinal tract and thus may help relieve constipation and other gastro-intestinal problems.

Estimates of current average dietary fiber intake for adults in the U.S. are 10 to 30 grams/day, with men averaging 19 and women 13 grams/day. An intake of up to 40 grams of fiber/day or 25 grams/1000 kcalories of food appears to be beneficial. A maximum intake of 50 grams would be a reasonable recommendation. A practical approach for most individuals is to establish the current level of intake and gradually increase it, with the goal of doubling the intake. Fiber supplementation is not encouraged.

Modest amounts of sucrose and other refined sugar may be acceptable for some individuals. Their use is contingent on the effects on blood glucose and lipids as well as maintenance of body weight. The statement concerning use of sucrose was not intended to encourage the use of foods high in refined sugars, but instead to assist persons with diabetes to use these foods more intelligently, if that is their choice. They need to be aware that portion sizes are usually small, how to substitute correctly for them in the meal plan, and that good times to use these foods are usually at meals when these foods appear to be absorbed more slowly, and before exercise. The advice to be careful of foods containing significant amounts of sugar is good nutritional advice for everyone, with or without diabetes.

Protein

The role of protein in the diet for persons with diabetes is unclear. Research is currently examining this question. Have the traditionally high protein diets recommended for persons with diabetes contributed to the development of renal disease? Until this issue is resolved, protein recommendations will be controversial. e Task Force Report states that Americans, in general, consume too much protein - usually two to three times the R.D.A. The intent of this recommendation is to encourage health professionals to begin thinking about de-emphasizing the role of protein in diabetes.

Fat and Cholesterol

Total fat should comprise less than 30 percent of the calories and dietary cholesterol should be less than 300 mg/day. The recommendation again is to individualize, the amount must be achievable within the context of the overall nutritional prescription, and the meal plan must be palatable.

Persons with diabetes have a high risk of cardiovascular disease (CVD). Abnormalities of plasma lipids and lipoprotein concentrations are also associated with greater CVD risk. Because of the established favorable effects of fat modified diets on lipids and lipoprotein concentration, the minimal requirement of a diet for persons with diabetes should be comparable to the American Heart Association (AHA) Phase 1 recommendations. All types of fats - saturated, ployunsaturated, and monounsaturated - should be proportionally reduced.

Alternative Sweeteners

The use of alternative sweeteners, both nutritive and non-nutritive, is thought to be acceptable in the management of diabetes. Persons should be cautioned concerning the caloric content of the nutritive sweeteners (e.g. fructose and sorbitol); to use a variety of sweeteners to dilute any potential risk; and to use moderation. Use of sweeteners should be evaluated in the context of the overall nutritional value of the diet.

Salt

It is recommended that sodium intake not exceed 3,000 mg/day. This recommendation endorses the AHA recommendations for sodium. The principal concern with sodium intake is for people with hypertension and those susceptible to its development. People with diabetes are frequently hypertensive, and control of hypertension is one of the first steps in the management of the micro-and macrovascular complications associated with diabetes. Caution is directed toward the patient who has problems with fluid volume or hypotension where salt intake may be beneficial.

Alcohol

Use of alcohol is not encouraged, but if it is an individual's choice to use alcohol, individuals need to know how to use it safely. Specific problems with the use of alcoholic beverages for persons with diabetes may occur, such as hypoglycemia, glycemic control, obesity, and hyperlipidemia.

If alcohol is consumed it should be in moderation. For individuals requiring exogenous insulin, occasional use of 2 equivalents of an alcoholic beverage can be consumed in addition to the meal plan. No food should be omitted because of the danger of alcohol-induced hypoglycemia and because alcohol does not require insulin to be metabolized. Occasional use was defined as not more than 2 equivalents once or twice a week. One equivalent (1/2 ounce of pure ehanol) is equal to the amount of alcohol, contained in 1.5 ounces of a distilled beverage (such as gin, bourbon, whiskey, vodka, dry brandy), 4 ounces of wine, or 12 ounces of beer. Dry wines and light beers contain less carbohydrate than sweet wines or regular beer and are better choices. Alcoholic beverages should always be consumed with food.

Alcohol is high in calories when weight is a concern, it is best substituted for fat exchanges because it is metabolized in a manner similar to fat (1 alcohol equivalent = 2 fat exchanges).

Vitamins and Minerals

There is no evidence that persons with diabetes have unique needs that warrant vitamin or mineral supplementation. Vitamin and mineral recommendations that apply to the general public also apply to persons with diabetes. Caution is expressed in regard to persons on very-low-calorie diets.

Education

Nutrition therapy and education must: (1) clearly differentiate nutrition goals for the different types of diabetes; (2) be related to the patient's cultural and economic status; and (3) be presented in ways that can be understood and implemented.

To teach minority patients about nutrition, educators much utilize educational techniques appropriate to the patient and his family. Single concept messages for diet instruction in NIDDM often promote learning and minimize failure. Nutrition information should be divided into sequenced manageable steps which can be individualized to the patient's setting. No single set of educational materials will be appropriate for all minority groups or situations. A variety of tools and techniques must be used by nutrition educators. These must relate to the foods of a particular group and utilize sound education strategies (7).

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Nutrition and Chronic Diseases: Discussion

Nelson, Maudine

I would like to know from Dr. Howard and Ms. Franz if there are any recent findings on diabetes and obesity that are unique to blacks.

Franz, Marion

Some investigators in Florida have recently identified a type of diabetes that occurs in black adolescents, and they have called it Type 1 1/2 - diabetes. The affected youths present with the typical symptoms of Type-1 diabetes, have a very high blood glucose value and they are ketotic. They are put on insulin but after a period of months or maybe years, they can discontinue use of insulin and at that point, seem to be more like Type-2 diabetics. This type of diabetes is so far found only in black youths and is currently under further investigation.

Howard, Lyn

I don't think there is anything specially new in relation to obesity. What is loud and clear about obesity is that the overwhelming prognosticator is your genetic input and you should choose your parents very carefully.

Grossman, E.M.

I would like to ask Dr. Howard why there is such a huge prevalence of obesity among poor black women as depicted in one of her slides? In other words, obesity is more common among the poor than the rich; it is more common in females than among the males, and it is more common among black females than their white counterparts. What are the reasons?

Howard, Lyn

Your statements, as depicted in my slides, are correct. One point I would like to mention which is a slight modification of that concept is the recent finding that in children, prevalence of obesity in the low socioeconomic group is actually lower in black children than in white children. That situation flip flops as they go into adolescence and adulthood, with the poor black women tending to have the highest incidence. I have to assume that the latter trend is due to some major social factors.

Nelson, Maudine

What is the current status of the theory that the ability to retain salt is a very striking feature of blacks, and may be related to the blacks' experience in Africa?

Grossman, E.M.

An article in the Lancet some two years ago, written by an anthropologist, pointed out the long history of high salt intake and salt trade by inhabitants of the west coast of Africa. Those tribes obtained salt by evaporation of sea water, and therefore were never in short supply of salt compared with blacks living in the salt-poor inland parts of Africa. No one as yet to my knowledge has examined the differential susceptibility of these coastal and inland Africans to hypertension.

Kinnard, M

Dr. Grossman, I believe you mentioned that one gram of calcium could over come the blood pressure raising effect of one gram of sodium?

Grossman, E.M.

That is a rough calculation that I made from Zemel's published data.

Kinnard, M.

Are there any contraindications since calcium can be deposited in the joints and soft tissues?

Grossman, E.M.

I am not advocating supra RDA amounts of calcium. As you know from many surveys, huge segments of the U.S. population do not achieve the RDA for calcium. Secondly, the RDA for calcium has recently been raised particularly for certain segments of the population. The only people who have an absolute contraindication to increasing their calcium load are calcium stone-farmers, and they are a tiny minority of the U.S. population. Because the stones are calcium oxalate stones, they have to limit their calcium intake as well as avoid spinach, rhubarb, and a whole bunch of other foods that are rich in oxalates. Lactose intolerance is also a relative contraindication.

Cagney, Christine

I would like to have some more information from Ms. Franz about the ongoing trials of diabetes control and complications by the N.I.H.? Are blacks and native Americans included in the trial and what is their percentage relative to the total sample size?

Franz, Marion

I don't think race is one of the major criteria being considered at the moment. The criteria so far are age, duration of types of initial complications, etc. I think that when the trial is completed, it would be very interesting to separate out the factor of race.

There are about 28 centers involved in the trial and as a matter of fact, Vanderbilt University in Nashville is one of the centers. Each center enrolls approximately 50 people in the trial and they are taken from their clinical populations. I believe each center has to have people between ages 15-19 years old or something like that.

Anonymous

I have a question for Dr. Howard. I must say I am really impressed by the data you have presented. Many of us are aware of this so-called reciprocal regulation in biochemical pathways. While biosynthetic reaction is taking place, reciprocal regulation of degradative reaction is occurring simultaneously. If this reciprocal regulation results in a failure, one might have a futile cycle. It appears to me that in the role of genetic factors in obesity, not much work has been done in terms of imbalance in reciprocal regulation. I recently came across an article that discussed ATPase for example in the erythrocytes. How much of this derangement in enzyme regulation, probably as a result of genetic inheritance, has to do with deposition of calories as a result of non wastage of energy?

Howard, Lyn

When we eat, there are energetic processes that are true for everybody. They relate to processing of the food and are part of obligatory thermogenesis which represents roughly two-thirds of the energy released at that point. In addition, and depending on what is eaten, there is a facultative thermogenesis. It is a response principally through the sympathetic nervous system of increased energy expenditure which probably can be altered in its regulation, and that accounts for about one-third of the dietary-induced thermogenesis. One of the questions being addressed in the literature is whether there are inter-subject differences in facultative thermogenesis. Probably the most important papers that relate to this question are some recent ones in the JCI which looked particularly at the differences in lean, obese and diabetic patients in terms of their facultative response to glucose loads. What the papers show in effect, is that when we measure the response to food, the results are very controversial in terms of whether we follow it for 2, 4, 6 or 24 hours. In fact, it has been well demonstrated that in the diabetic, thermic response is altered by the preceding day's meal. So if you look at the 24-hour data which is what many people do, you can show that in fact, the persons who are beginning to develop glucose intolerance and insulin resistance, initially have defective facultative thermogenesis. They are not burning that glucose at the same rate. However, if you use insulin clamp studies so that you are pushing the glucose into their cells at the normal rate, they have normal facultative thermogenesis. If you then block their sympathetic nervous system with beta-propranolol, you can show that their ability to turn on their sympathetic nervous system is the same. Yes, there are differences between the lean and the obese which can be demonstrated in response to their energy expenditure from food, but they are probably not of the degree of importance we thought they were two years ago.

Anonymous

I don't believe you have really answered my question. I was looking for more specific examples: Glycogen synthesis and fatty acid synthesis for example are reciprocally regulated and if their is a derangement, you create a futile cycle.

Howard, Lyn

Facultative thermogenesis is this futile cycle you are referring to. It is the turning on, if you like, of inefficient use through the sympathetic nervous system. Now whether that occurs through brown adipose tissue, isometric contraction of muscles, or through specific up and down regulation via glycolytic mechanisms, is not yet precisely known. Clearly, that component is facultative thermogenesis. If you take a normal rat and make it overeat, it gains some weight but it's heat production increases dramatically. So, the rat has an incredible ability to stay some what in energy balance by increasing facultative themogenesis. This is not a major factor in humans who are in energy imbalance.

Fleshood, H.L.

Several speakers have indicated that major improvements in health will result from our ability to make positive changes in our lifestyles. How well are these issues being addressed in our medical schools? Second, is there any evidence that we pay as much attention to the very obese as we do to the quick fixes like surgery and other things?

Franz, Marion

It is certainly my impression that the medical schools are paying increasing attention to these issues as the public becomes more aware of them. I also think that as health professionals, our lifestyles must serve as a good model for the people we are trying to influence. Many of our medical centers are now concerned about the importance of wellness. In recent past, the traditional medical approach has been focused primarily on illness.

Grossman, E.M.

A serious joke among physicians is about a patient who went to his physician with a very bad cold. The doctor said, "I want you to stand in the rain for about 4 hours and get chilled." The patient was horrified and said that he would get pneumonia. To this the doctor retorted, "that we can cure."

My education as a physician as well as that of generations of physicians has unfortunately been to cure diseases. Proper nutrition is one important aspect of disease prevention, and an ounce of prevention is worth no less than a pound of cure. The exciting fields in medicine for tomorrow will be how much disease we can prevent, and what we are trying to do here in this workshop is to define the role of nutrition in preventive medicine.

About a year and a half ago, I participated in something called a focus group. We assembled two groups of physicians in Stamford, Connecticut to talk about calcium and osteoporosis. When the moderators were talking about osteoporosis, the response of most of the physicians was that they did not know what the fuss was all about since they felt they knew how to handle bone fractures. The point is that the whole orientation of physicians for generations has been to wait until the disease occurs and then try to cure it. This little speech is just to indicate that I agree with you on the importance of teaching medical students to be preventive physicians, and my hat is off to Meharry Medical College because that is what they are trying to do at the College's Center for Nutrition.

Rucker, Chris

The American Dietetic Association recognizes that a growing body of scientific evidence supports the positive relationship between consumption of plant-based diets and prevention of certain diseases. Marion Franz, in light of that and your discussion on diabetes, I would like you to comment on Dr. James Anderson's research on this disease, and the fact that he utilizes almost a total plant diet of 50 grams of fiber and also an hour of exercise per day to treat insulin-dependent diabetics, and gets about two-thirds of the patients off insulin.

Franz, Marion

James Anderson is a proponent of a high carbohydrate - high fiber diet. A major criticism of his research approach is that he does not use any control group, thus making it difficult to isolate the benefits attributable specifically to the fiber, high carbohydrate, low fat and weight loss.

Suskind, R.M.

We had a very interesting study going on at the Children's Hospital in New Orleans. We were looking at obese children aged 8 years through adolescence. Putting them on protein-sparing modified diets as out-patients, they lost weight safely and effectively, and at the same time, improved in terms of their body image and self-image. And it is really quite dramatic to see what I think we all intuitively feel about children as they lose weight, particularly the positive changes in their performance in school.

One other comment refers to our observation on five obese children who presented with insulin-dependent diabetes. These children, when placed on protein sparing diets, lost weight and were off insulin. The interesting observation was that on a follow up in these children, they no longer required insulin even though their weight reduction was not maintained.

Bernard, L.J.

I have been looking around the room to see if there is anyone from our department of psychiatry. Some of my psychiatry colleagues have made an observation with regard to obese black women. These psychiatrists contend that much of the obesity reflects chronic depression in the women who are poor and with relatively little expectations from living. In response to that depression, they turn to food.

I want to thank the panel and the audience. I think it has been a stimulating morning.

FOURTH SCIENTIFIC SESSION

Nutrition - Infection - Immunity Axis

CHAIRPERSON:

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Interaction of Nutrition - Infection-Immunity

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A well nourished status is required to offer maximal resistance to infections. Every infection, even though mild or subclinical has an adverse effect on the nutritional status of the child (1). The negative effect depends on the nature and severity of the acute infection, the previous nutritional state of the child, and on the diet during and immediately following the infectious episode. The nutritional damage caused by repeated infections can be cumulative if the child does not have time to recover from each bout of infections, that is, repeated infections cause progressive depletion of essential nutrients which, in malnourished children, can have serious consequences. It is estimated that, throughout the world, roughly 100 million children under the age of five years of age are moderately or severely malnourished (2). Protein energy malnutrition (PEM) is not just a problem of developing countries, but also of developed countries. In affluent societies the incidence of PEM is high in children affected by such chronic disease states as congenital heart disease, hepatic, and renal diseases. These children require a higher caloric intake than normal. It is therefore important to diagnose malnutrition before it becomes life-threatening. In moderately or severely malnourished children the most frequent effect of infection is impaired physical growth and development. In such cases, the diet is insufficient for normal growth. The consequences of acute infectious episodes, which may be unimportant in well-nourished children, can be catastrophic in the malnourished child.

Evaluation of Nutritional Status

Height-for age and weight-for-height are useful indices, together with a careful physical examination, to determine a child's nutritional status. The child with decreased weight-for-height is wasted or acutely malnourished, while the child with decreased height-for-age is stunted or chronically malnourished. According to Waterlow (3), children with weight-for-height in the range of 80-90% are classified as being acutely malnourished, Grade 1; children in the 70-80% range as Grade 2; and children below 70% as Grade 3. Children who are 90-95%, 85-80%, and less than 85% of height-for-age are classified as being chronically malnourished or stunted, grades 1, 2, and 3 respectively (3).

To prevent severe malnutrition, every child at risk who is younger than five, and below the 25th percentile for height should be carefully

*Speaker

assessed by anthropometric and biochemical measurements to evaluate his/her nutritional status. In addition to weight and height, anthropometric measurements of head circumference, triceps skinfold should be performed. Head circumference is a useful indicator of cerebral mass and of brain growth (4). A head circumference below the 5th percentile for age is often associated with poor cognitive development. Triceps skinfold has the advantage of changing very little between ages one and four years (5), the period in which risk of malnutrition is greatest. This parameter correlates strongly with real body weight, but is independent of weight gain due to ascites or constipation. The skinfold measurement correlates well with total body fat content and indirect indicators of total body fat include electrical conductivity and ultrasound (6). However, the skinfold measurement is easier and less expensive to obtain. It is advisable to take two or more skinfold measurements (at the triceps and subscapular points) because the mean of these parameters provides a more accurate estimate of total body fat. These parameters are compared with standard NCHS (7) because genetic differences do not significantly affect the growth potential during the early years of life. However, the height and weight growth rates in two to four year olds are slower in developing countries than in children from more affluent societies. This is undoubtedly due to the exceedingly high number of repeated infections in these already malnourished children (8).

In addition to anthropometric parameters, biochemical and nutritional intake data are essential for evaluating the child's nutritional status. Where clinically indicated, the biochemical parameters include data on visceral proteins, vitamins, minerals and trace elements. Such parameters require invasive procedures which are very expensive. However, some baseline measurements as well as indicators of certain nutrient deficiencies should always be obtained.

A general hematologic and biochemical assessment of nutritional status should include: a complete blood count, albumin, transferrin, prealbumin, iron, transferrin saturation, ferritin, vitamin A, carotene, vitamin D (alkaline phosphatase), vitamin E, vitamin K (or prothrombin time), folate, thiamine, riboflavin, pyridoxine, vitamin B12, vitamin C, and zinc (9).

Nutritional intake can be assessed using 24-hour recall, in which the patient is asked to recall all foods consumed within the past 24-hours, for 3-4 successive days.

Features of Malnutrition

Severe malnutrition can manifest itself as kwashiorkor or marasmus. Both can occur as primary disease states secondary to other disease states.

Kwashiorkor is caused by acute protein loss or deprivation. Children affected by kwashiorkor have edema, skin lesions, hair changes, apathy, anorexia, an enlarged fatty liver, decreased serum albumin and abundant subcutaneous adipose tissue. The children usually recover promptly with a high protein diet. Marasmus is caused by severe chronic malnutrition due to a deficiency of several nutrients essential for caloric intake. These children are usually younger than those affected by kwashiorkor, and present with growth retardation, weight loss, muscle atrophy and loss of subcutaneous tissue. In summary, the substantial difference between the two entities is that marasmus is the result of a chronic adaptation to a very low caloric intake, while kwashiorkor reflects an acute protein loss (10) leading to a decrease in visceral protein synthesis.

Children with primary or secondary PEM show nutritional deficits which can include protein, calories, vitamins and minerals. Consequently, children affected severely by PEM show a wide variety of problems in biochemical and physiologic functioning. Such alterations affect responsiveness to infection. All organ systems are altered in children with PEM. One of the most important to be affected is the gastrointestinal tract. PEM reduces velocity of cell proliferation, migration of the enterocytes, and secretion of digestive enzymes both from pancreas and from the mucosal cells of the intestinal wall. These children show an altered metabolism of bile salts with an increase in the deoxycholate/cholate ratio and consequently an impaired ability to form cells with the resultant malabsorption of fat (11). Such alterations, together with the gastroenteritis often observed in these children, cause malabsorption of a large number of nutrients which further worsens the nutritional state of the child. In these children the heart rate is reduced, together with a decrease in cardiac output and peripheral blood flow. Very often varying grades of anemia may complicate the clinical picture. The renal system is also very severely affected with a decrease in the rate of glomerular filtration as well as a decline in tubular function.

Metabolic and biochemical alterations affect almost all aspects of homeostasis in children with PEM (12). Protein metabolism has been studied extensively. One of the most widely used indicators of altered protein metabolism is reduced circulating plasma albumin, transferrin and retinol-binding protein. In contrast, the globulin fraction of plasma protein is not as significantly affected. In general, total protein turnover in children with PEM is reduced, but such turnover is maintained at almost normal levels in liver. The total plasma level of amino acids is reduced by about 50% in children with severe PEM. This reduction is more severe in the essential amino acid fraction while the non essential amino acid fraction is normal or occasionally increased. The low protein turnover leads to considerable nitrogen conservation demonstrated by reduced levels of both plasma and urinary urea. One of the most important indicators of altered lipid metabolism is a fatty infiltrated liver in children with kwashiorkor. Such an alteration is due to a secondary decreased apoprotein synthesis (12).

Carbohydrate metabolic alterations may be related to alteration in the levels of insulin and corticosteroids. Although liver glycogen stores as well as fasting blood sugar levels are reduced, the efficient use of gluconeogenesis provides glucose homeostasis to a degree compatible with survival. As for mineral metabolism, a decreased total body potassium has been reported. This may be related to muscle breakdown, or impaired activity of the ATP-requiring sodium-potassium pump, which may also explain the intracellular accumulation of sodium (12). The metabolism of zinc, iron, copper and magnesium is also impaired in children with protein calorie malnutrition.

Both extracellular and intracellular total body water is increased. The pathogenesis of edema in children with PEM is not completely clear, although water and sodium retentions are related to hypoproteinemia as well as to multiple hormonal and mineral changes (12). Children with PEM show a number of alterations in several endocrine parameters. These children, although stunted, frequently show very high levels of growth hormone, which are inversely related to albumin levels (12, 13).

Insulin levels are also very low, even after glucose infusion or after stimulation with glucagon. Low insulin levels lead to reduced protein synthesis in the muscle. These children show elevated plasma levels of 17-hydroxycorticosterioids and cortisol. Cortisol diurnal rhythm is also altered. The increased cortisol level favors hepatic synthesis of plasma proteins such as albumin (14).

All of these alterations affect the basal metabolic rate (BMR) in malnourished children. Children with PEM are hypothermic, bradycardic, physically inactive and demonstrate reduced emotional responsiveness. When the nutritional status of the patient improves the BMR also returns to normal (15, 16). Malnourished children are also unable to maintain normal body temperature and fail to respond to both cold and heat stress (17, 18, 19). These alterations are further reduced by intercurrent infectious disease (20).

Effects of Infections on Nutritional Status

While it is true that a malnourished child is highly susceptible to infection, it is also true that repeated infections can lead to a state of both acute and chronic undernutrition. Every infection may influence different aspects of the nutritional state of the child. Every mild infection may cause some degree of anorexia and reduced food intake, in part, due to the release of Interleukin-1 (21). In addition, during the course of an acute infection, it is a common practice to substitute low calorie and protein liquids for solid food (22). Each systemic infection, be it viral, such as measles (23), or bacterial (24) can reduce the absorption of principal nutrients. Absorption is further reduced if the infection involves the GI system (25, 26). Consequently, if an ill child has very limited stores of nutrients because of pre-existing malnutrition, even a short period of decreased food intake, when the energy requirements are increased, can cause a severe nutritional insult. Carbohydrate stores are inadequate to meet these greater needs (27) and lipid and protein stores are used (28). Indeed, the nature, duration, and severity of the infectious disease can affect lipid metabolism. This is particularly evident in gram-negative infections (29), in which defective serum lipid clearance results in extreme hypertriglyceridemia (30). During infectious diseases, the utilization of ketones is impaired; consequently, endogenous proteins are used to meet caloric requirements.

Every acute systemic and localized infection of viral, bacterial, rickettzial, protozoic, and helmintic origin has a negative effect on the protein metabolism. Infections cause not only an increased utilization of plasma amino acids for the synthesis of acute phase reactants, anabolic repair, and stress response (31,32), but also an increased hepatic metabolism of amino acids for gluconeogenesis. The amino acids for gluconeogenesis come from muscle with the nitrogen being excreted in the urine as urea. Powanda has calculated a daily loss of approximately 0.6 g protein/kg of body weight in individuals with acute infections of different etiology with a mean duration of 162 days (33). In the case of nitrogen loss not compensated for by an increased protein intake, as in the case in malnourished children, there is a further notable protein depletion and the appearance of kwashiorkor-like syndromes. During infection numerous changes occur in a number of minerals (34), such as decreased serum levels of iron and zinc and an increase in serum copper. Although chronically reduced levels of iron, zinc and copper increase the frequency of infections, acute reduction in the level of plasma iron and zinc can be considered a component of the acute phase response. Indeed, reduced available iron is a major host defense mechanism (35) because low serum iron levels, particularly associated with fever, severely limit bacterial growth (36, 37). Iron uptake is mediated by unsaturated lactoferrin, an intracellular iron binding protein released from neutrophils (38) which binds serum iron; the complex non-protein is cleared from circulation, resulting in hypoferritinemia. In the end, the sequestered iron appears as hemosiderin which cannot be used. Decreased iron levels are probably mediated by Interleukin-1 which induces the release of lactoferrin from neutrophils (39,40). Another essential nutrient for bacterial repletion is zinc, which is a normal component of the bacterial membrane and ribosomes and is incorporated into several bacterial proteins (41). During infection, it may be due to Interleukin-1 that host develops hypozincemia because of the increased production of metallothionine, a metal binding protein synthesized as an intracellular acute phase protein reactant (42, 43, 44). Interleukin-1 also seems to effect an increase in plasma levels of copper, accelerating the hepatic synthesis of ceruloplasmin. Ceruloplasmin oxidizes ferrous iron and may increase the efficiency of iron utilization for hemoglobin synthesis to compensate for the decrease in iron availability.

Increased metabolism and nutritional requirements may continue well into convalescence from the infectious disease, resulting in elevated requirements for a number of nutrients for a prolonged period of time.

Effect of Malnutrition on the Immune Response

The effects of infection on the nutritional state of the host are extremely serious when they occur in malnourished children. Malnutrition by itself increases the frequency and severity of the infections as a result of decreased specific and non-specific defense functions. Children with PEM also show alterations in the integrity of anatomic barriers such as the epidermal and mucosal membranes. Because of the altered gastrointestinal tract permeability, macromolecules from the digestive process can pass from the intestinal lumen into blood, increasing the formation of serum antibodies to common food antigens (45).

In addition, there is a decreased secretion of lysozyme and gastric acid. Decreased intestinal secretion of hydrochloric acid causes an increased gastrointestinal pH which allows replication and passage into the small intestine of pathogens responsible for the increased episodes of diarrhea. Finally, the development of the normal febrile response is altered in these children and this, too, may impair the specific immune response (37,46). In children with PEM several specific defense mechanisms are impaired such as the cell mediated immune response (47), the humoral immune response (48), the phagocytic and killing function of leukocytes (49), and the complement system (50). Several of these functions are impaired by a decreased production of Interleukin-1 by macrophages (51).

The Cell Mediated Immune Response (CMI)

The CMI is the most compromised and perhaps the most researched defense mechanism in children with PEM. It has been well documented at autopsy that children with kwashiorkor have severe thymic atrophy. More recently, a decrease in the size of spleen, lymphoid tissue and tonsils has been demonstrated (52). On histologic examination significant degeneration of the Hassel's corpuscles and decreased numbers of small lymphocytes in the thymus have been noted (52). Paracortical depletion and reduced germinal centers in the lymphoid nodes have been reported (53). Such lesions are more severe in children with kwashiorkor than in children with marasmus (53). The most practical way to evaluate CMI in children with PEM *in vivo* is by intradermal skin testing and, *in vitro*, by the enumeration of T-lymphocytes and by antigenic and mitogenic stimulation of isolated lymphocytes. To demonstrate an adequate cell-mediated immuneresponse there must be healthy, circulating, thymus-derived T-lymphocytes which can be sensitized to an antigen. Later, the sensitized lymphocyte must be able to recognize the antigen and respond with the release of lymphokines.

One of us (RMS), as well as many other investigators, have researched this field and demonstrated that children affected by PEM, before nutritional therapy (supplementation), are unable to develop a normal inflammatory response, whereas children who are well nourished can respond positively (10). To evaluate the recall phenomenon, children with PEM have been sensitized with DNFB at different times during nutritional therapy. Only 20% of children sensitized on the first day of admission to the hospital were able to recall the sensitization after 72 days, while 87% of those sensitized on day 56 of hospitalization and consequently with a better nutritional status, recalled the challenge on day 72. The results of this study suggest that most children with severe PEM do not have circulating lymphocytes which can be sensitized.

Testing *in vivo* shows that most children affected by PEM are unable to recall previous sensitization to a foreign antigen. However, because of the depressed inflammatory response, it is difficult to determine whether the decreased skin test reactivity is secondary to a depressed recall phenomenon or to a depressed inflammatory response itself.

Cell-mediated immunity can be evaluated *in vitro* by the absolute lymphocyte count. Most children with severe PEM have a lymphocyte count of less than $2500/mm_3$. However, this is an extremely poor prognostic indicator (53). The most frequently used test *in vitro* to evaluate cell-mediated immune function is peripheral blood lymphocyte transformation in response to stimulation with mitogens, with resulting non-specific activation of lymphocyte proliferation. The most frequently used antigen are PHA and Concanavallin-A. Children with PEM show a lower mean proliferation index on hospital admission than on discharge. Likewise, blast cell transformation is significantly lower on admission than on discharge. The improvement in nutritional status causes normalization of T-cell response to mitogens (10,54). The decreased number of rosette-forming T-cells is often related to an increase in the number of null cells. This could be the consequence of an impaired production of thymic hormone, such as is found in serum obtained from children with PEM (55).

In addition, children affected by PEM show a pronounced reduction in T4 helper cells and a moderate reduction in T8 cytotoxic, suppressor cells (56). These and other alterations of CMI in children with PEM can be explained by a significantly reduced Interleukin-1 in the serum of these patients (57,58). Several investigators have demonstrated that the serum of children with PEM contains factors capable of inhibiting such immunological responses as *in vitro* lymphocyte transformation (45). This may be related to the decreased responsiveness of T-cells to mitogens and to the formation of rosettes (59,60).

Such factors could be C-reactive protein (CPR), which is highly elevated in children with PEM, pheto-proteins (61), or perhaps others. However, the mechanism of these processes still awaits investigation.

The Humoral Immune System

The function of the B-lymphocyte system is evaluated by counting the number of B-cells, measuring circulating immunoglobulins, measuring the antibody response to antigenic stimulation, and by studying secretory immunoglobulins.

B-Cell Enumeration

The B-cell number in children with PEM is normal or increased. This could be due to increased exposure to various pathogens (62) or to a depressed T suppressor cell population, causing uncontrolled non-specific antibody production (63).

Serum Immunoglobulins

Most children with PEM have either normal or elevated circulating IgA, IgM and IgG levels (64,65). Occasionally, a malnourished child, without a concomitant or recent infection, has decreased serum IgG, IgA and IgM levels (48). Generally, the increased values of IgA and IgM, return to normal levels as the nutritional status of the child improves (10), very probably because the incidence of infectious episodes in these children concurrently diminishes. Increased serum IgG and IgE have also been observed in malnourished children (66,10). High IgE levels may be secondary to an increased frequency of parasitic infections or an increased hypersensitivity to a variety of allergens perhaps due to defective suppressor T-cell function (48). However, this does not indicate that the humoral immune system of malnourished children is normal and efficient.

To evaluate the competency of the system one must determine its response to a foreign antigen challenge. Generally, the response to a specific antigen is determined by the nature and dose of antigen and the degree of malnutrition present in the child. Responses to diptheria and typhoid antigen are reduced (67), while the specific antibody response to tetanus and polio virus is normal (59, 60). The response to measles virus and measles vaccination is variable, being reported as either normal or decreased (59, 60, 67).

The altered function of the humoral immune system may be due to non-immunological factors such as insufficient availability of essential amino acids required for synthesis of several antibodies. Other factors may be high levels of serum corticosteroids that may interfere with antibody-binding capacity. Immunological defects can be related to a disorder in antibody production. At least five different soluble factors are involved in production, differentiation and activation of antibodies and may be altered in PEM. These factors are B-cell growth factor, B-cell differentiating factor, colony stimulating factor, I-2, and IL-1. In children affected by PEM, Interleukin-A levels are notably reduced and this could be related to altered IgG functioning. However, additional research is needed to clarify this process. Finally, it has recently been shown that a-interferon increases immunoglobulin synthesis (69), but interferon production is lower than normal in children with PEM (54). As the nutritional status of the malnourished child improves and protein intake increases, the humoral immune response improves as does the cellular immune response.

Secretory IgA System

Children affected by PEM often present with a high incidence of respiratory and GI system infections. Therefore, many investigators have evaluated the secretory immunoglobulin system. Studies done in Chiang Mai, Thailand have demonstrated that secretory IgA levels in the respiratory tract are decreased. However, after 10-12 weeks of nutritional therapy secretory IgA returns to normal levels (10). Chandra has demonstrated a similar reduction in secretory IgA levels in the respiratory system as well as in the GI tract. It is not yet clear if the damage is in IgA producing mucosal plasma cells and/or immune cell populations which produce the secretory component (70).

The secretory IgA reduced levels, both in respiratory and GI systems, may explain the decreased immune response to some viral vaccines (70, 71), the increased incidence of septicemia by gram-negative bacteria, and the high incidence of serum antibodies to common food antigens found in these children.

The Phagocyte Function

Phagocytic function in PEM has been extensively examined and can be divided into different phases: a) chemotaxis, b) engulfment, c) phagocytic vacuole formation and degranulation, d) microbial killing and e) concomitant metabolic changes (10). The number of circulating phagocytic cells is normal in children with PEM.

Generally, phagocytic function in vivo is studied utilizing the Rebuck skin window technique. Children with PEM show normal neutrophil, but depressed mononuclear cell chemotaxis (10). Douglas et al, using in vitro techniques found an initial delay in the migratory response of cells from malnourished children but, within 3 hours, the values reached control value (72). Phagocytosis of various particles by polymorphonuclear leukocytes or monocytes from children with PEM is normal. Electron microscopic studies of polymorphonuclear leukocytes from children with kwashiorkor using S. aureus, E. coli, and Candida Albicans have demonstrated no apparent qualitative abnormality in vacuole formation or degranulation (49).

The phagocytes of malnourished children show increased baseline levels of hexosemonophosphate shunt activity and a normal resting reduction of NBT dye. These biochemical changes indicate relatively normal polymorphonuclear leukocyte biochemistry (10). Thus, the polymorphonuclear leukocyte in malnourished children appears to respond appropriately to dermal injury. It can phagocytoze bacteria adequately, and can kill bacteria adequately in the majority of patients with PEM, although many enzymes are altered (10).

The Complement System

Activation of the complement system leads to production of complement fragments which are involved in host defenses, including viral neutralization, chemotaxis of polymorphonuclear leukocytes, monocytes and eosinophils, opsonization of fungi, endotoxin inactivation, lysis of virus infected cells, and bacteriolysis (50).

In children with PEM the complement system, as well as other immune responses, are impaired. These children have decreased levels of total hemolytic activity (10). Some investigators have found increased consumption of complement components by infections (54, 73).

We also evaluated the serum level of complement proteins in classical as well as C3 activating systems in children with PEM on admission and after nutritional therapy with different initiates of calories and protein. Although the importance of infections in complement utilization cannot be ruled out, the serum complement levels are probably reduced secondary to impaired synthesis as a result of the decreased protein and calorie intake. All of the children with PEM on admission showed significantly lower than normal levels for eight of nine complement components. Children on a higher protein-calorie intake showed improved recovery, reaching normal complement levels sooner than those with PEM on lower protein and calorie diets. More precisely, children with lower protein intakes, did not reach normal complement levels, therefore demonstrating the importance of a high protein and calorie intake in the resynthesis of complement components.

The reduced function of the complement system, suggested by the decreased protein levels, has been confirmed by the impaired hemolytic complement activity in children with PEM. However, hemolytic complement activity was reduced only in children with severely acute PEM. Children with mild or moderate malnutrition showed no such alteration.

Complement activating factors could be partly responsible for the impaired hemolytic complement activity. Such factors(s) could be circulating immune complexes, endotoxin, macroglobulins, c-esterase inhibitors and C3 and C6 inhibitors. We found 30% of children with PEM had endotoxemia (10).

Summary

Children with PEM have increased morbidity and mortality associated with infection and a depressed immune response. Several investigators have demonstrated depressed *in vivo* and in vitro cell mediated immunity in children with PEM. Although circulating immunoglobulins are elevated in PEM, the ability of the malnourished child to respond to certain antigenic stimuli is compromised by the child's nutritional status. In contrast to the other systems, PEM does not appear to depress the chemotactic and phagocytic ability of polymorphonuclear leukocytes. Some investigators have observed depressed polymorphonuclear leukocyte killing activity in malnourished children, while others have found depressed killing function only in selected patients. Individual complement proteins and hemolytic complement activity in children with PEM are significantly depressed. Investigators have also noted anticomplementary activity and circulating endotoxin in the serum of some malnourished children.

As a result of the depressed cellular immune, humoral immune, and complement systems, the child with PEM is at more risk of developing recurrent, overwhelming infection.

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Nutritional Implications of Acquired Immune Defficiency Syndrome

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Introduction

Since early 1981 when the Acquired Immune Deficiency Syndrome (AIDS) was initially described, over 42,000 cases have been reported to the Centers for Disease Control (CDC). The first case definition for AIDS by CDC was "a reliably diagnosed disease that is at least moderately indicative of an underlying cellular immunodeficiency in a person who has no known cause of underlying cellular immunodeficiency or any other underlying reduced resistance reported to be associated with that disease" (1). A revision of the first case definition in June 1985 expanded the diagnosis of AIDS in certain diseases if the individual has a positive serologic or virologic test for the AIDS virus (2). Recently a third revision of the CDC surveillance definition for AIDS expanded the reporting criteria further to include illness characterized by "indicator" diseases (3). A classification system established by CDC in May 1986 is useful in understanding the progression of AIDS from the acute infection period to asymptomatic infection to manisfestations of life threatening opportunistic infectious diseases or cancers (Table 1) (4). This classification system is also helpful in determining when nutritional problems occur and to what extent. The Human Immunodeficiency Virus (HIV), formerly known as Human T-lymphotropic virus type III (HTLV-III), lymphadenopathy- associated virus (LAV), and AIDS-associated virus (ARV), causes the immunological abnormalities seen in this disease. The syndrome which follows this infection is characterized by lymphopenia, elevated immunoglobins, reduced T-helper cell number, inverted T-helper cell to T-suppressor cell ratio and decreased monocyte response to antigens (5). The virus can be detected by either culturing the virus or identification of antigens or HIV-specific antibodies (6). The two most common tests are ELISA (enzyme-linked immunosorbent assay) or Western Blot. Both detect HIV-specific antibodies produced by the infected person's immune system (6). People at risk for infection by HIV are those who engage in high risk behavior such as homosexual, bisexual, or heterosexual contact with an infected person, multiple sexual partners of unknown status, needle sharing intravenous drug abuse and use of or contact with contaminated blood products.

Common symptoms seen in individuals on clinical evaluation can be: malaise, easy fatique, fever, and/or night sweats, unexplained weight loss, chronic diarrhea, shortness of breath, lymphadenopathy, lesions of the skin or mucous membranes and signs of opportunistic infections (7). Common infections seen in patients with AIDS are pneumocystis carinii pneumonia (PCP), cytomegalovirus (CMV), mycobacterium avium-intracellularae (MAI), candida, herpes, cryptosporidiosis.

Nutrition is a vital part of the care for the patient with AIDS. Sometimes the number and severity of infections and other medical problems require multiple changes in nutritional care plans. Some of the current nutrition therapies used in other diseases, especially cancer, can be adapted for this patient population.

Nutritional Assessment

Nutritional assessment of patients with HIV infection or AIDS is similar to that of other hospitalized patients. A good criterion of nutritional and metabolic assessment is by Blackburn et al (8). Monitoring parameters of anthropometrics, laboratory and nutritional/diet prescription can determine the nutritional status and progress of a patient with AIDS.

Monitoring anthropometrics over time is well suited for this patient population as they usually have numerous clinic visits and admissions. When determining a percent of weight, the usual ideal body weight formula is not valid (9). A more valid measurement is the use of the patient's usual or pre-illness weight in calculation of percent of ideal weight (9). Calculating percent weight change is a good tool in assessing significance or severity of weight loss (8). Percent weight change is important to assess as significant weight loss of 10% is part of the symptomatology of the constitutional diesease (4).

To estimate muscle mass and fat stores, arm circumference, and triceps skin folds can be measured and compared to standards (8). With successive admissions, these measurements can be compared to previous admission measurements to determine if changes in muscle mass and fat stores are occuring.

In assessing visceral protein, monitoring laboratory data on serum albumin, total iron binding capacity and serum transferrin can help determine changes (8). The use of total lymphocyte count and delayed hypersensitivity in this population is not a valid assessment tool as they would be a measure of immune function in this disease. A possible way to use these parameters would be a carefully monitored nutrition support study with serial measurements of total lymphocyte count and delayed hypersenstivity skin testing.

Calculation of caloric needs is done using the Basal Energy Expenditure (BEE) equation (8). This equation takes into account the parameters of weight, height, and age. When calculated the BEE is multiplied by a stress factor for either enteral or parenteral, maintenance or anabolism (8). Protein requirements may be based on grams per kilogram body weight (10) or kilocalorie to nitrogen ratio of at least 150:1 (11).

Fever, a frequent symptom in AIDS, affects energy expenditure. There is an increase in metabolic rate of 13 percent for each degree celsius and 7.2 percent for each degree fahrenheit above normal (12). Fever, the response to infection, also produces other nutritional considerations such as anorexia, nausea, vomiting, altered production and utilization of metabolic fuels, and negative balance of electrolytes (13).

Besides anthropometircs and laboratory data, assessment of dietary intake prior to admission and during admission is helpful information. In the medical admission evaluation there are usually references to nausea and vomiting, esophagitis, anorexia, chronic diarrhea and dehydration. These admitting symptoms will give an idea of oral intake of fluids and solid food before admission. Evaluation of food and fluid intake during admission can be followed by daily calorie counts and nursing flowsheets compared to calculated nutritional needs.

Nutritional Assessment Studies

Even though there is limited informaion in nutrition texts on nutrition and AIDS, there are several papers in the current literature.

A study by Kotler et al on body composition in patients with AIDS demonstrated that there was significant depletion of body cell mass (14). Measurements used in this study were total body potassium, fat, total body and extracellular water volumes as well as serum retinol binding protein level, iron binding capacity and albumin. These measurements were repeated over time to detect changes in body mass (14).

Another study on nutritional status and body weight was done by O'Sullivan et al (15). In their review of medical records of patients with AIDS they noted an average weight loss of 16% from admission weight (15). In a similar nutritional status study by Garcia et al, it was determined that there was a mean weight loss of 11.8 + 7.6 kilograms from pre-illness weight to weight at death (9).

Assessment of iron binding capacity, albumin, midarm circumference in individuals with AIDS was done as part of study on diarrhea and weight loss by Kotler et al (16). In assessing the effect of AIDS on gastrointestinal function and structure, patients were found to have significantly lower values of iron binding capacity, albumin, and midarm circumference than the controls (16). The resulting diagnosis on all patients in this study was malnutrition (16).

Chlebowski gives the following as possible explanations for weight loss and malnutrition seen in AIDS: malabsorption, simple starvation, or altered metabolism resulting in alterations in organ function and immune status, increasing morbidity and mortality (17). Chlebowski notes that the mechanism of malnutrition and its treatment in solid tumor patients is not directly applicable to the malnutrition seen in the AIDS patient (17).

Assessment of certain vitamins and minerals is also important as they are involved in immune function. A study on selenium in AIDS documents a deficiency of this important mineral (18). It was found that blood selenium levels were significantly diminished in AIDS patients (18). However the exact mechanism for this was unknown as it occured independently of presence or absence of malabsorption and had no correlation to serum albumin (18).

Besides malnutrition affecting organ function, mortality and morbidity, it also affects immune function. Immunological changes in AIDS are also seen in protein energy malnutrition (19). There is some hypothesis that due to similarities between the two, that poor nutritional status contributes to the immunodeficient state (19,20,21). There is also some discussion that malnutrition may be a contributaory cause for some cases of AIDS seen in the Haitian population (22,23).

Providing Nutritional Care: Asymptomatic Period

Nutritional complications present themselves at different stages of the disease. In the asymptomatic period of HIV infection, few if any nutritional problems occur. However nutrition is important for asymptomatic individuals in maintaining health and weight. Encouragement of a balanced diet, good eating habits, and a daily vitamin and mineral supplement should be provided at this time.

Constitutional Disease

As an individual progresses through the classification system defined by CDC (Table 1) (4), nutritional problems develop. Occurence of nutritional problems, their severity and duration differ between individuals.

The most common time for nutritional problems to start occuring is in what is called "Constiutional Disease" period, formerly known as AIDS-Related Complex (ARC). During this time an individual may experience constant fever, involuntary weight loss of greater than 10% and diarrhea persisting for more than a month (4). Nutrition intervention is determined on a individual basis by encouraging adequate intake to meet increased caloric needs. Routinely, a high protein high calorie diet with the use of complete supplements is prescribed. When diarrhea is a persistent problem, the diet prescription can be low fiber, low lactose, low fat with use of lactose free supplements. Sometimes modular or elemental supplements can help maintain adequate intake.

Opportunistic Infections and Malignancies

When an individual is diagnosed with an opportunistic infection or malignancy, he is classified into subgroup C and D. At this time multiple nutritional problems occur. Nutritional problems may be related to the actual disease process or treatment.

Many times, pulmonary infections such as PCP or Kaposi sarcoma cause nutritional problems. Due to dyspnea and coughing, adequate oral intake of food can be inhibited (24). Small meals, nutritious snacks and complete liquid supplements can be used to augment oral intake.

When opportunistic infections occur in the gastrointestinal tract, nutritional care is of great importance. Opportunistic infections or malignancies can occur throughout the gastrointestinal tract (25). In the oral cavity, candida (thrush), herpetic lesions, Kaposi sarcoma and hairy leukoplakia occur. It has been observed that candidiasis is prevalent in all groups while Kaposi sarcoma is more frequently seen in homosexual/bisexual individuals with AIDS than in drug abusers (26). Esophageal infections can also inhibit food intake. Infections commonly found in the esophagus in AIDS are candida and cytomegalovirus. The most frequent oral and esophageal infection is candidiasis which causes dysphagia and odynophagia (25).

When the small and large bowels become infected, diarrhea and malabsorption frequently become difficult problems to overcome from a nutritional standpoint. Opportunistic infections which invade both small and large bowels may be parasitic, bacterial, or viral in nature. The bowel may also have Kaposi sarcoma lesions and diffuse undifferentiated lymphoma (27). The actual structural damage of these opportunistic infections vary. Cytomegalovirus (CMV) may cause small bowel injury due to vasculitis by the virus infection of endothelial cells . When CMV is in the colon the organism is found in the mesenchyme of the lamina propria or muscularis mucosa producing a colitis (27). Cryptosporidia organisms are found in the microvillus epithelial brush border and are associated with endoscopic erosions (27) and produce a cholera-like diarrhea for several months (28). Mycobacterium avium intracellulare (MAI) produces blunted villi with foamy macrophages similar to Whipple's disease (29). The Kaposi sarcoma involves the muscularis propria and mucosa, and may cause ulceration (27).

Diarrhea caused by infectious organisms can be difficult to overcome. The volume of diarrhea may be 10-30 bowel movements per day or 10-15 liters per day (9). In some cases diarrhea can be caused by nonpathogenic protozoa such as entamoeba coli (30). Diarrhea from these types of entamoebas can be moderate to severe but usually responds to metronidazole therapy with some documented relapses (30).

An enteropathy has been identified in AIDS where bacterial or parasitic infections are not detected as a cause for diarrhea (16, 25). On histological examination there seems to be a pathologic process occuring in the lamina propria of the small intestine and colon (16). In an observation by Brinson, hypoalbuminemia in AIDS patients can be considered a cause for diarrhea when infectious etiology is not found (31). In both conditions, reduction of D-xylose absorption occured with the diarrhea (16, 31). In some instances especially cryptosporidiosis infection, diarrhea may be secretory in nature and does not respond to dietary intervention or NPO status (9, 32).

Malabsorption may differ depending on part of the bowel involved. If the large colon is infected and colitis is present, water and electolytes may be malabsorbed. If the small bowel is infected, absorptive function can be assessed by D-xylose test or fecal fat analysis (33). Nutrients malabsorbed with diarrhea are monosaccharides (glucose, xylose), disaccharides (lactose), nitrogen, fat, vitamin B12, folate, vitamin A, minerals and trace elements (34).

Dietary intervention at this time is individualized to patient's tolerance and what part of the GI system is affected. When oral lesions or infections are present, a soft, semi-soft or liquid diet is used. Use of high protein high calorie complete supplements should be encouraged to insure adequate calorie and protein intake. When the esophagus has lesions, ulcerations or is inflammed, a soft or liquid diet and use of complete supplements are implemented. If esophagitis is severe enough a gastrostomy tubefeeding or hyperalimentation may be alternative form of nutrition support. If gastritis is present due to infections, a bland diet with elimination of untolerated foods may help. Supplement use depends on individual tolerance. The high osmolarity of complete supplements may cause gastrointestinal upsets; isotonic or modular formulas are sometimes tolerated better.

Dietary intervention when the small and large bowels are involved causing diarrhea is often unsuccessful. Diet modification, however, can assist in alleviating the severity of symptoms. A lactose restricted, low fiber, low fat diet can be helpful. Supplements usually tolerated are isotonic lactose free or modular formulas. Use of an elemental formula is some- times successful if the patient can tolerate it via the oral route. When ever diarrhea is a problem, adequate fluid intake is encouraged to prevent dehydration. Again, it is important to recognize that if the diarrhea is secretory no dietary intervention will be helpful (9, 32). At this point hyperalimentation is an option (9, 32). Increased risk of infection from administration of parenteral nutrition in the immune compromised patient is a concern. However, if proper technique and care are followed the catheter site should pose no significant problem.

Other organs which may become involved in AIDS are the kidneys and liver. Abnormal tests of liver function and hepatomegaly may indicate hepatic involvement (35). Infections and/or malignancies found in the liver in AIDS may be cryptosporidia, cytomegalovirus, mycobacterium, hepatitis B, kaposi sarcoma and lymphoma (35). Renal dysfunction particularly proteinuria, may be caused by multiple infections, volume depletion, or use of potentially nephrotoxic drugs (36). Dietary changes in protein, sodium or potassium are done when fluid status, liver or renal function deteriorate. However, the patient usually is not eating well at this point and it is difficult to get the patient to eat enough to meet the dietary needs.

Possible Nutrition Role in AIDS

One area where there has been some speculation about is the role nutrition may play in the initiation or development of AIDS. Already discussed is how malnutrition may play a role in exacerbating the immunoincompetence in AIDS. A hypothesis by Archer and Glinsmann goes further with this idea by suggesting that repeated intestinal infections cause malabsorption which leads to malnutrition and then AIDS (37). The suggested treatment for this is rigorous parenteral nutrient repletion, antibiotics aimed at eliminating intestinal infections and immunologic boosting therapies (37). A deficiency of essential fatty acids has been hypothesized as a predisposing factor for AIDS (38). Begin and Das propose that polyunsaturated fatty acids, gamma-linolenic and eicosapentaenoic acids could be natural endogenous agents against AIDS (39). Due to their ability to destroy enveloped viruses these fatty acids may prevent spread of viral infection (39).

Home Care

Home health care is common for patients with AIDS. It is important that nutrition is part of the home care referral. Description of diet prescription, supplements and how many times per day the patient is to receive them need to be addressed. If discharge to a home care agency is made with hyperalimentation or tubefeeding, appropriate information on strength, rate and toleration should be given in the referral.

One area that needs to be addressed during discharge planning is patient's actual physical strength and financial resources. The physical ability to actually buy and prepare meals may be impaired. Due to severe muscle atrophy, profound fatigue, and neurological damage (40, 41), sometimes patients are unable to go grocery shopping and prepare meals. Support groups, friends or family members can help in buying and preparing meals and assisting with feeding if needed. Care attendants provided by the home care agency can also assist in these tasks. If financial help is needed to buy foods, social services can assist with applications to state or federal agencies. Food stamp program may also help buy foods. Some support groups and relief organizations have food pantries which may be another source of food. A social worker can assist the patient in acquiring these kinds of services.

Special precautions in kitchen care and food preparation are not required. Maintaining a clean food preparation area and utensils is essential. Cleaning can be done with a bleach solution of 1:10 dilution. When preparing fruit and vegetables they need to be washed thoroughly. Meats need to be cooked and maintained at appropriate temperatures. Encourage patients to store left over foods for a minimum of three days, then dispose of them. Warn patients against eating unpasturized milk products, raw eggs and food left out for extended period of time.

Drug-Nutrient Interactions

Many times, drug therapies in the treatment of AIDS can cause side effects which inhibit adequate nutrient intake and utilization. This is especially true during extended hospitalization where many different drug therapies are utilized.

As in cancer, chemotherapy is a culprit for causing inadequate nutrient intake. The most common chemotherapeutic agents used for Kaposi sarcoma are: bleomycin, vinblastine, vincristine, and adriamycin. Nutritional complications experienced with these agents are stomatitis, loss of appetite, nausea, vomiting and diarrhea. Some patients have reported taste alterations. Chemotherapeutic agents used in lymphoma treatment, methotrexate and procarbazine, also produce similar nutritional problems especially nausea and vomiting.

Certain antibiotics produce nutritional problems. Aminoglycosides and trimethoprin/ sulfamethoxazole may precipitate severe nausea and vomiting causing the patient to eat inadequately through out the antibiotic treatment.

Amphotericin B, besides nausea and vomiting, may cause metallic taste and anorexia. Rifampin may cause sore mouth, loss of appetite and possibly, diarrhea. Pyrimethamine has side effects of vomiting, loss of appetite and tenderness of tongue at high dosages. Metronidazole besides nausea and vomiting, may cause dry mouth, metallic taste and possibly, diarrhea. Isoniazid, an antitubercular drug, causes disruption in folate, niacin, pyridoxine and B12 metabolism along with epigastric pain, anorexia and dry mouth.

Pentamidine is used as a alternative therapy in Pneumocystis carinni pneumonia and has the potential of altering the metabolism of glucose. It has been observed to induce hypoglycemia which may continue after therapy ends or develop several days after pentamidine has been stopped (42). The mechanism by which pentamidine causes hypoglycemia is not known but it has been proposed that it is toxic to the beta islet cells of the pancreas (42).

Research drugs used in the treatment of AIDS present unknown nutritional problems. In administration of DFMO (Difluoromethylornithine) there could be some nausea, vomiting and diarrhea. With DHPG (9-1, 3 dihydroxy-2- propoxymethyl guanine) possible mild nausea and vomiting can occur. DFMO is used in treatment of cryptosporidiosis and DHPG in treatment of cytomegalovirus. Other investigational drugs such as ribavirin and AZT (azidothymidine) have limited data on possible nutritional complications. Some patients during nutrition counseling sessions have reported taste alterations while on AZT, especially with cooked foods. Ribavirin may have the gastrointestinal effects of nausea, vomiting and diarrhea. Ampligen, another investigational drug, reportedly causes only mild nausea as a possible side effect.

Conclusion

The disease AIDS demands a large amount of care especially nutrition care. The nutritional problems which develop as a result of impaired immunity, opportunistic infections or malignacy and drug treatments require individualized counseling and diet prescription. As significant weight loss is part of the symptomatology of the constitutional disease, nutrition counseling in maintaining weight must be a part of HIV care. Financial and social situations of patients with AIDS must also be assessed in planning for patient to follow diet criptions at home. More research as well as clinical experience needs to be achieved to help determine the best method for nutritional assessment, intervention and evaluation.

Table 1

Summary of Classification for Human Immunedeficiency Virus Disease*

Group I. Acute Infection: patients with transient signs and symptoms of HIV infection.

Group II. Asymptomatic Infection: patients must have had no previous signs or symptoms that led to classification in Groups III or IV.

Group III. Persistent Generalized Lymphadenopathy (PGL): palpable lymphadenopathy at two or more extra-inguinal sites persistig for more than 3 months.

Group IV. Other Disease: clinical manisfestation may designate patients to one or more subgroups below whether they are minimally symptomatic or severly ill

- Subgroup A. Constitutional Disease: one or more of the following: fever persisting more than 1 month, involuntary weight loss of greater than 10% of baseline, or diarrhea persisting more than 1 month.
- Subgroup B. Neurologic Disease: dementia, myelopathy, peripheral neuropathy.
- Subgroup C. Secondary Infectious Diseases: diagnosis of an infectious disease at least moderately indicative of a defect in cell-mediated immunity.
  - Category C-1: symptomatic or invasive disease due to one of 12 infectious diseases listed in surveillance definition of AIDS.
  - Category C-2: symptomatic or invasive disease due to one of six other specified secondary infectious diseases.
- Subgroup D. Secondary Cancers: diagnosis of one or more kinds of cancer known to be associated with HIV infection.
- Subgroup E. Other conditions in HIV Infection: presence of other clinical findings of diseases, not classified above, that may be attributed to HIV.

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*Adapted from Centers for Disease Control, MMWR 35 (20):p335, 1986.

Table 2

Gastrointestinal infectious organisms in AIDS.

Type	Organism	Drug Therapy
Parasite	giardia lamblia	metronidazole
	entamoeba histolytica	metronidazole
	crytosporidium	DFMO*
	isospora belli	bactrim
	non-pathogenic amoeba	metronidazole
Bacteria	shigella	bactrim/ampicillin
	salmonella	bactrim/ampicillin
	mycobacterium avium-intercellularae	possible anti-TB medication
	campylobacter	erythromycin
Virus	cytomegalovirus	DHPG**, spiramycin
	herpes simplex	acyclovir

-Difluoromethylornithine

** 9-(1,3-dihydroxy-2-propoxymethyl) guanine

Adapted from M.E. Garcia et al, Nutrition in Clinical Practice 2 (3):p109, 1987.

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Nutrition and Cancer Among United States Minority Populations

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Following are abbreviations used in the text and tabular material: C.I.=confidence interval, BMI=body mass index=Quetelet Index={(weight (kg)/height (cm)^2)*10,000), kg=kilogram, g=gram, RDA=recommended dietary allowance, NHANES=National Health and Nutrition Evaluation Survey, TSNS= Ten State Nutrition Survey, NFCS=Nationwide Food Consumption Survey, NK=Natural Killer, PUFA=polyunsaturated fatty acid, PMN=polymorphonuclear neutrophil, PG=prostaglandin, SHBG=sex hormone binding globulin.

*Paper presented by JRH.

Introduction

The topic of nutrition and cancer is a vast and often confusing one. Despite many gaps in our knowledge, we are called upon to draw inferences and make judgements based on existing data and to choose promising options for future research.

Often our clues concerning etiologic relationships and our motivation for future study are based on differences in disease rates between culturally dissimilar populations. For many cancers, whose dietary connections are suggested by findings from various types of studies, United States black/white differences in incidence and mortality rates are striking. The goal of this paper will be to focus on specific cancer sites for which available epidemiologic evidence suggests marked black/white differences. We will discuss specific dietary factors that may contribute to these differences. We will also discuss possible mechanisms of action, with special emphasis on immunology. We will conclude by listing suggestions for future study and making recommendations for dietary modifications that are consistent with decreasing the risk for all major causes of mortality in the United States.

Sources of Evidence.

Evidence for a link between nutrition and cancer comes from a variety of sources. These include:

-ecological studies; usually these are based on cross-sectional comparisons of aggregate food disappearance data with disease rates across countries or other geopolitical units.

-trend studies; these may include changes in disease rates with migration or changes in rates of disease within a defined geographical area over time.

- laboratory animal investigations
- in vitro experiments [cellular metabolism]
- quasi-experimental or observational studies in humans which focus on biological markers of nutritional exposure or cancer outcome or both

-epidemiologic studies, under which could be subsumed:

- case control studies
- follow-up or prospective studies
- clinical or intervention trials.

Though these categories of studies are not always separate and distinct, they represent the major categories of scientific endeavor from which evidence is obtained. The sequence in which we listed the types of evidence was deliberate. The strength of evidence should increase in proceeding from cross-country comparisons, where we rely on crude estimates of aggregate exposure and outcome, to nutritional intervention studies in humans, in which we can randomly allocate dietary regimens and measure and control for potential confounders in analysis. Despite the fact that bona fide epidemiological studies should furnish the most conclusive form of evidence for diet/cancer relationships, on balance, the strongest available evidence implicating nutrition in various types of cancer comes from what are generally regarded as the less definitive types of studies. There are several methodological problems related specifically to the study of nutritional effects that may severely compromise conventional epidemiological investigations. We have discussed these problems at length elsewhere (1) and will touch upon them later in this paper.

While neither the ecological studies nor the laboratory experiments allow us to make strong inferences about causal relationships, they do provide us with material upon which hypotheses are formulated and they help us to describe possible mechanisms of action. Along with available evidence from case-control and follow-up studies, they help us to formulate reasonable views regarding the role of diet in cancers of various sites. Many of the individual relationships are of special relevance to black Americans, whose rates for certain sites differ markedly from those of whites.

Specific Cancer Sites Possibly Related To Diet

Our discussion will attempt to focus on cancer sites whose rates either currently differ between whites and minorities or are exhibiting race-differential rates of change over time.

Prior to launching an ecological or analytical study one usually observes cancer incidence or mortality rates that differ drastically across geographic units. These differences in cancer site-specific rates usually follow economic or development gradients, often with the highest rates in the more affluent Western countries.

Figures 1, 2, and 3 illustrate the differences in cancer rates for the colon; bronchus, lung and trachea; and prostate between populations in Africa, the Caribbean, and North America. Rates for breast and endometrial cancer among females reveal a similar pattern [2]. Cervical cancer, whose incidence is thought to be determined largely by transmissible agents, varies much less drastically across these groups. Except for cancers such as primary hepatocellular carcinoma, Burkitts Lymphoma, and stomach cancer, which are generally related to defects in sanitation or unique patterns of food preparation, blacks living in the less developed countries have much lower cancer incidence and mortality than their counterparts in the countries of the developed world. Though there are some striking differences in cancer rates between races within countries of the West, in general, rate differences between blacks in Africa and blacks in North America are much larger.

It has been several decades since Dr. Denis Burkitt, working in Africa, noticed a very low incidence of diverticular disease and colon cancer among black populations eating foods very high in fiber [3]. Since then, diets containing low dietary fiber and high fat have been related to cancers of various sites.

Differences in mortality rates between the affluent and poor countries are also very large, parallelling the race-, sex-, and site-specific differences observed for cancer incidence in the registries' areas [4,5]. In general, when one pursues an ecological lead, mortality data are used in analyses. Unlike incidence data which are available from specific cancer registries' catchment areas, aggregate mortality data are available on a country-wide basis for many of the countries of the world. Food disappearance data are also available at this level of aggregation.

Ecological studies done by Wynder and colleagues [6], Armstrong and Doll [7], and Carroll and Khor [8] could be interpreted to implicate specific dietary factors in cancers of many sites. Most of the striking differences in cancer rates, however, exist in places not only distinct for their dietary practices but for their racial composition as well. Until studies of migrants established that immigrants take on the cancer rates of their new environment [9-11], it was suggested that differences in cancer rates, sometimes of more than a full order of magnitude, were due to genetic factors. While some cancers may have strong genetic determinants, for most of the common cancers environmental factors are the primary determinants. This is not to say that genetic factors are unimportant. Within any cultural context we can observe familial aggregation of certain cancers such as those of the colon and breast. Genetic factors which protect from or predispose toward cancer may interact with environmental factors. We know some race and sex-specific metabolic differences that may relate to cancer risk. For example, blacks appear to metabolize compounds in cigarette smoke differently from whites [12]. This may help to explain differences in cancer rates for sites that are conventionally thought to be tobacco-related but for which simple smoking rates do not sufficiently account. Still, for most disease processes, race probably functions more as a cultural than a biological descriptor [13].

Based on worldwide ecological evidence, sometimes corroborated by evidence from other sources, the strongest accumulation of evidence for a diet/cancer link is with cancers of the digestive system, followed by endocrine-related organs, the organs of the upper aero-digestive tract, and finally, assorted other sites [14-16]. Rather than repeat these excellent reviews on the current status of the diet/cancer field, we will go on to discuss specific U.S. non-white/white differences in disease rates and exposure characteristics that we feel warrant further consideration. A summary of cancers whose race-specific rates or changes in rates vary markedly are listed in Tables 1-3 along with potential nutritional factors of relevance.

Cancer Sites For Which U.S. Non-White and White Rates Differ

Our primary source of data for race-specific cancer mortality rates in the United States provides the rates dichotomized by whites versus non-whites [17,18]. Though the rates for blacks tend to dominate the overall non-white rates, it is important to note that there are differences between black and other non-white rates and that blacks are not one homogeneous group. Despite this, much can be gleaned from the simple white/non-white rate comparisons.

Within the United States, for all sites of the digestive system combined, rates for non-whites are slightly higher than those of whites. Aggregating all sites of the digestive system, however, masks some important differences, as we show in Figure 4. Among non-whites of both sexes, stomach cancer mortality exceeds that of whites by about 60 to 75% [17,18]. It is known that for stomach cancer, methods of food preservation such as curing and pickling and diets low in vitamin C are positively associated with incidence [19]. As we shall see, these are all factors that differ between black and white subpopulations in the United States.

Esophageal cancer, which we group with the aero-digestive cancers typically associated with tobacco and alcohol, has a much higher mortality among blacks, with rates three or more times higher in both sexes. Colon cancer, whose primary dietary association is with fiber and fat, appears to be about 10-20% lower in blacks. It should be noted, however, that rates were much lower for blacks over past decades, that they have been increasing, and they are apparently continuing to catch up. As with stomach cancer where a single diet-related subsite dominates when conditions favor cancer [19], cancer of the left-sided or descending colon is the subsite that dominated in circumstances of high incidence and for which a dietary connection is hypothesized [16]. Despite the strong suggestion that dietary factors determine colon cancer risk, the disease also tends to show familial aggregation.

In males, the rate of the main endocrine-related cancer, i.e., prostate, drastically differs between the races (see Figure 5). It has been suggested that dietary factors, especially high fat and low zinc, may play an important role [20]. As we shall point out, diets of blacks, in general, appear to be slightly higher in fat and moderately lower in zinc than the diet of whites.

Among non-white females, we would expect endometrial and breast cancer rates to be lower than in whites due to the protective effects of early and multiple pregnancies. Breast cancer mortality is only slightly lower while the endometrial cancer mortality rate is actually one and one half times as high [17,18]. Because of the strong relationship between obesity and endometrial cancer [14,16,21] and the higher rate of obesity in the American black population, this is not too surprising. Recent evidence from Seattle indicates that breast cancer incidence may be rising among blacks [22]. This is consistent with a trend over time toward relatively higher breast cancer mortality in non-whites [17,18]. Cervical cancer, known to be related to sexual practices, and recently found to be positively associated with smoking [23], is much higher in non-white than white women [17,18]. One study has also shown that low vitamin C intake is related to uterine cervical dysplasia [24].

Some of the most striking race-specific differences in cancer rates occur in the organs of the upper aero-digestive tract. Laryngeal and oral cavity cancer rates are, on average, 60% higher in non-white versus white males. While both sites are related to alcohol and tobacco use, several nutrient deficiencies such as iron and B vitamins have also been implicated. Esophageal cancer rates differ between the races drastically and the differences have developed relatively recently [17,18].

Although a greater proportion of blacks than whites smoke cigarettes, they tend to be lighter smokers, as shown in Figure 6. This argues against cigarette smoking or alcohol intake, which is actually higher in whites, as sufficient explanation for rate differences. Our observation that tobacco-related variables do not fully account for between-country differences in lung cancer mortality motivated an ecological study on dietary fat and lung cancer which showed a strong effect of dietary fat on lung cancer after accounting for important covariates [25].

While dietary fat may, in fact, have nothing to do with lung cancer, such a relationship could help to explain some of the lung cancer rate differences between blacks and whites in the United States and the striking differences in rates between males in the U.S. and males in Japan who tend to smoke much more, eat much less fat, and have lower lung cancer rates [4,5]. Two epidemiologic studies have implicated dietary fat or cholesterol in lung cancer [26,27] and a few experimental studies on laboratory animals have been carried out which lend support to a possible relationship between dietary fat and lung cancer to illustrate that nutrition may play an important role in explaining cancer rate differences, even in cancers with established strong risk factors.

In general, cancer rates for non-whites have been rising faster than those for whites. While non-whites have lagged behind in the cancer sites associated with "Westernization," such as colon and breast, they are now quickly approaching the rates of whites [17,18,22]. Where rates have been higher, such as for esophageal and prostate cancer, they have tended to continue to diverge. Where rates have been dropping for both races, proportional decreases have been lower in non-whites such as for stomach and cervical cancer.

Tables 1, 2, and 3 summarize the sites for which non-white/white cancer rate differences are largest or for which race-specific changes over time are largest. Most plausible nutritional factors of interest are listed alongside the cancer site. It must be noted that many of these dietary relationships are speculative and should be interpreted as interesting foci for future research.

Cancer Survival

In general, blacks have poorer cancer survival than whites. To some extent this is due to factors related to stage at diagnosis such as nodal involvement which, in turn, relates to access to medical care and quality of follow-up care. Even after accounting for stage, however, there is a survival advantage to being white, as Figures 7 and 8 indicate for cancers of the breast and prostate, respectively. In our own study of breast cancer survival, we have found overweight to be the strongest predictor of decreased survival aside from stage of disease at diagnosis (Herbert JR et al unpublished findings). This is consistent with the findings of other researchers [30,31] and the known increased prevalence of obesity and poorer survival among blacks.

Specific Dietary factors

In this section we will highlight what can be learned from the data available on black/white differences in nutritional status. Specific nutrients that have been implicated either as protective or risk promoting agents in cancer causation are listed in Tables 1,2, and 3. In addition to specific nutrients and food types, we will consider race differences in rates of obesity and in alcohol consumption. Assessment of body habitus is considered a nutritional sub-specialty and energy balance, on which body mass is ultimately based, is determined to a large, but varying extent by total dietary intake. Alcohol consumption is important to consider because of its role both as a co-carcinogen [32] and activator of the mixed function oxidases, and as a potential contributor to total caloric intake.

What we know about the possible role of specific nutrients, patterns of eating behavior, and overweight or obesity in cancer is derived from a wide array of sources. The quality and strength of evidence vary markedly among individual dietary components, and across the cancer sites of interest. A large shortcoming of dietary survey data is their dependence on self-reports by respondents. Unlike assessment of current diet or recall of past diet, which may be associated with large sources of random or systematic error [33,34], total body weight and height are relatively easy to measure with moderate to good precision and accuracy [35], and past weight may be recalled with a reasonable level of success [36].

Data on the prevalence of obesity in the U.S. are available from the first and the second National Health and Nutrition Examination Surveys (NHANES I and II) which obtained direct measures of height and weight. In addition, the National Health Interview Survey [37,38] and the Hypertension Detection and Follow-up Program [39] collected self-reported heights and weights. NHANES I [40], conducted between 1971-1975, and NHANES II [41], conducted between 1976-1980 were designed as nationwide probability samples of the civilian non-institutionalized U.S. population. In neither of the two NHANES surveys were significant black/white differences of relative ponderosity detected among adult males [42]. Data from NHANES II did show a tendency toward overweightness increasing with age among males 35 to 55 years old [43].

In contrast, BMI {weight(kg)/height(cm)^2 * 10000} for black females in NHANES II were significantly higher than for white females across all age groups 12-74 years, with larger increments of difference at higher ages [42]. Overweightness among black females rose steadily from 30% at age 25 to 60% at age 45, with black rates roughly double those of white women between the ages of 45-55. Multivariate analysis showed that obesity is independently predicted by race, after controlling for age, sex, and socio-economic status [43]. These findings are consistent with the prevalence of obesity found among women aged 40-60 years in the TSNS [44] where the maximum rate of obesity was reported at 49.7per cent. In a survey conducted on 200 black households in Mississippi [45], the percentage of 48.7% (for the 40-60 age range) was considerably less than the maximum

In her compilation of age-adjusted overweight prevalence rates from five major nationwide surveys, Kumanyika [46] has determined that black women are nearly twice as likely as white women to be classified as overweight. Relative weight for height measured do not distinguish between the fat and fat-free, or lean body mass, tissue components. Anthropometric data, however indicate that black females accumulate larger excesses of fat tissue than do white females. Higher tricep skinfolds were reported for black in TSNS and in NHANES I [46]. Triceps skinfold data are thought to provide a marker of fat in the extremity whereas subscapular skinfolds measure trunk fat. Adult-onset obesity is characterized by excessive trunk fat, and is known to predispose individuals to greater risks for development of chronic diseases, such as diabetes, hypertension, and cardiovascular disease. In their analysis of NHANES I data, Blair et al [47] found larger black/white differences for subscapular skinfolds than for triceps skinfolds among women 30-59 years of age (26.7 mm vs 19.4 mm and 26.2 mm vs 24.2mm, respectively). This pattern of excess trunk fat distribution is consistent with data on skinfold measurements in children, and appears to emerge in late adolescence [46].

The consistency of these patterns in U.S. black women suggests that the elevated risk profiles for certain cancers might, in part, be attributed to their increased prevalence of obesity. Elevated body mass index (BMI) has been most strongly associated with endometrial cancer [21] for which non-white rates are much higher [17,18] and which may help to explain the lack of protective effect usually attributed to early and multiple pregnancies. A recent study of breast cancer by Schatzkin et al [48] found that in postmenopausal women obesity (defined as BMI> 30) was associated with a relative risk estimate of 2.5 (95% C.I.: 1.5-4.4), compared with lean women. Poorer survival for cancers of the breast, uterus, cervix, and endometrium among black women suggests that obesity may potentiate a differential effect on mortality among the races. The obesity-survival relationship is certainly subject to confounding by socio-economic factors which affect access to, quality of, and continuity of medical care.

When patterns of caloric intake are assessed relative to differences in ponderosity, a marked discrepancy is found among black females who tend to report significantly lower caloric intakes per kilogram body weight than whites. In NHANES II, black females from 21 to 74 years of age consumed between 94 and 115 calories less per day than their leaner white counterparts [42]. Black males sampled in NHANES II ingested fewer calories per kilogram body weight across all age groups , with significant differences noted for those 21 to 65 years of age. Koh in 1984 [40] and more recent results from the 1985 U.S.D.A. Nationwide Food Consumption Survey (NFCS) [50,51] have similarly reported significant racial differences in energy intake. While the contrast in energy nutriture is generally less marked across female age groups, overall lower energy consumption for blacks of both sexes has been consistently reported in a number of surveys [44,52-55]. This illustrates a potential difficulty in obtaining accurate survey data. In the *Future Studies* section we will discuss the weaknesses inherent in dietary assessment tools currently in use in epidemiologic research.

The apparent discrepancy between food energy consumption data and the prevalence of obesity in black females may be at least partly explained by the crudeness of the dietary assessment methods used. It may also suggest that the development of obesity is not only a function of total caloric intake, and points to the possibility of a difference in the assimilation of foodstuffs (e.g. blunted thermogenic effect of food) and differences in levels of physical activity. Metabolic energy expenditure and physical activity, however, cannot be feasibly measured in large-scale surveys. If, in fact, the survey data reveal a true difference in energy intakes, a question is raised about the adequacy of food selections in meeting minimum dietary requirements for the macronutrients and accompanying vitamins and minerals.

Since certain minority subgroups may be at risk of low total energy intake, it is necessary to consider potential problems with nutrient densities, or the relative proportion of specific nutrients that compromise dietary intake. In a study by Koh [49], it was found that blacks consumed 56g of fat, whereas whites consumed 66g. These amounts reflected 38% of total energy consumed by blacks, compared with 33% by whites. Moreover, the types of fats used most frequently varied noteably. Total fats derived from vegetable sources were 34% of total fat calories for blacks and 37% for whites. This finding is consistent with the U.S.D.A. food purchasing profiles in which Salathe et al [56] reported greater expenditures by blacks on pork, poultry, and eggs than on complex carbohydrates, such as cereals, fresh fruits and vegetables. In contrast, whites showed greated consumption of plant foods which are generally lower in saturated fat content. Expressed in comparison to non-hydrogenated vegetable oil, blacks consumed much more hydrogenated fats than did whites. Blacks ate nearly two times as much hydrogenated fat as vegetable oil (22% vs 12%) compared with whites (19% vs 28%) [49].

Protein is the single nutrient most frequently reported at adequate levels of intake for both blacks and whites [49,57]. This is significant in that many high protein foods (meat, cheese, etc) are also high in fat. Food frequency data from NHANES I [58] showed a higher percentage of blacks eating meat and poultry two times or more per day. Eggs were also consumed more frequently by blacks of both sexes.

In both NHANES, protein consumption by whites exceeded that of blacks across all adult age groups. This finding was similarly noted by Koh [49] who reported a ratio of animal protein to vegetable protein intake of 3.25 for blacks, compared with 2.72 for whites. These data, taken together with the household food consumption profiles, point to a larger proportion of total energy needs potentially met by animal protein sources, including chicken, processed meats, pork, and eggs. In contrast, the balance of calories consumed by whites is evidently derived from a greater intake of carbohydrates. White males aged 12 to 74 years in NHANES II ingested significantly more carbohydrates than black males. Similarly, amounts of carbohydrate consumed (per kg body weight) by black females aged 45 to 65 years were significantly lower than for white females [42].

Utilizing 3-day dietary intake data from the 1977-78 U.S.D.A. NFCS [55], Cronin et al [59] noted that blacks consumed fewer fruits and vegetables per day than did whites (2.3 fruit and vegetable items per day compared with 2.9). NHANES I indicated a higher percentage of blacks consuming these food items once a day. A greater percentage of whites, however, reported eating fruits and vegetables twice a day. Both the Cronin and NHANES analyses revealed few reported differences in intake of cereals among whites and blacks. With respect to simple sugars, however, blacks were found to consume both candy bars [60] and sweetened beverages [59] more frequently.

Diets low in energy will also have an increased likelihood of being deficient in micronutrients. This was reflected in the U.S.D.A. NFCS of 1977 [55] and 1985 [50,51] where daily mean intakes of vitamin B6, calcium and magnesium achieved less than 81% of the 1980 recommended dietary allowances (R.D.A.) among blacks. These nutrient profiles were all notably higher among the white comparison groups, for whom reported energy intakes are generally higher. Black men achieved 87% of the R.D.A. value for vitamin E, and 88% for zinc in 1985, compared with 99% and 95% among white men. Both black men and women had exceedingly low levels of mean folacin intake (61% and 47% respectively, vs 77% and 51% among white men and women).

Findings from NHANES I [61] highlighted that mean calcium intakes consistently fall below the study standard for black women between the ages of 20 and 74 years. Among black females an average of 71% of the sample aged 18 to 44 years were found to have low calcium intake compared with 52% of white females in the same age group. Among black males the largest proportions at risk for low calcium intake was 45%, in the 45-74 year age strata compared with 25% among white males. Inadequate levels of calcium and vitamin D intake have frequently been reported for black population groups, and are largely attributed to low intake of milk and diary products [44,54,59,62,63].

Whereas mean levels of vitamins A, C, thiamine, and riboflavin were generally adequate across all population groups sampled in NHANES I, mean iron intakes fell below standard for all groups. Among black males, proportions of low intake ranged from 15% to 49% among 20-74 year olds. For black females aged 18 to 54 years an average of 93% of intakes were below standard. Black boys and girls of 1 to 3 years of age showed the highest percentage of individuals (95%) with inadequate levels of iron intake [52].

In contrast to NHANES I, a recent report [64] on mean vitamin C intake by race from NHANES II indicates the higher prevalence of more marginal levels of intake than previously noted. Low intakes were most noted for the 30-54 year age group across all race and sex groups, with the greatest inadequacies noted for all groups below the poverty index (56% and 50% for white males and females, compared with 49%, 52% for blacks).

In evaluating the source of vitamin C most frequently selected by blacks, Block [64] has raised an important distinction between naturally occurring ascorbic acid as ingested from fruits and vegetables and the synthetic varieties used to fortify fruit drinks. Since cancers of the digestive system have been more closely ssociated with vitamin C-containing foods than with the isolated nutrient [65,66], Block's distinction points to an important difference in the primary sources of vitamin C ingested by white and black populations. It is the complement of other naturally occurring compounds in vitamin C-containing foods (e.g, carotenoids, aromatic istohiocyanates, vitamin E, selenium, and bioflavonoids) that may potentially augument some of the free radical quenching effects of ascorbic acid. Thus, population groups meeting their nutrient requirements from fortified foodstuffs may not be receiving the full health benefits as those conferred by freshly-derived sources of the vitamin. In population-ranked profiles of the major food contributors of vitamin C, fortified fruit juices were reported as second only to orange juice for blacks aged 19-54. Among blacks 55-74 years this vitamin C source was ranked item number 4, compared with item number 11 for the same white age category.

Block has hypothesized that vitamin C intake (per kg body weight) is inversely proportional to the relative differences noted in black and white mortality from cancer of the stomach, esophagus, prostate, and cervix (white females < black females < white males < black males). This observation strengthens previous reports of the anti-tumor promoting properties of vitamin C [14,24].

The dietary fiber values computed from NHANES II reveal relative consumption patterns among blacks and whites that resemble the same inverse trend noted above for vitamin C, with black males and females lagging behind their white counterparts. The population-ranked profiles of foods contributing dietary fiber revealed that vegetables are a leading source in the U.S. population [67]. This finding highlights the enhanced risk of poor blacks in procuring adequate quantities of dietary fiber and vitamin C from their daily food selections.

The specific nutrients we have discussed are ones that figure prominently in the diet/cancer controversy. Next, we will consider specific ways in which the effects of these nutrients are expressed.

Mechanisms of Action

Immune Function

Our growing interest in the role of cell-mediated immunity in carcinogenesis and the inclusion of this paper within the topic of nutrition and immunity, motivates our discussion of the role of nutrition and immunity in cancer.

Immune surveillance of neoplasms has become an area of increasing interest in cancer research. Since the immune surveillance hypothesis was first suggested in 1909 [68], there has been substantial evidence that immunodeficiency diseases are associated with a higher incidence of a variety of cancers including lymphomas and leukemias [69]. In addition, immunosuppressive agents have been associated with increased tumorigenesis [70].

A component of the immune system, natural killer (NK) cells, have been recognized for their role in tumor identification and destruction [71,72]. It is widely known that the ability of the immune system to defend against viruses/bacteria can be severely hampered by certain nutritional deficiences and possibly some excesses. In the same manner, immune system defense against neoplasms may be negatively affected by inadequate or poor nutrition [73].

Nutrition mainly affects cell-mediated immunity, whose components play a major role in early tumor surveillance and in defense against viruses. Generally, malnourished individuals have fewer circulating T-cells [74]. The immunological effects of specific nutrients such as vitamin B6, pantothenic acid, folic acid [75] and B12 [76] are also well known. Deficiency in copper can reduce functioning of the thymus reticuloendothelial system and antibody producing cells [77]. Iron and zinc deficiencies are associated with multiple abnormalities in cell and humoral immunity [77]. Iron deficiency can result in marked changes in cytotoxic reactions, secretory IgA levels, lymphokine production and phagocytosis [77]. Zinc deficiency is linked to impaired granulocyte mobility [78], depressed T-cell number and T-cell mitogen response, T-helper function, and NK cell function [79,80]. Zinc deficient laboratory animals have shown a depressed T-killer cytotoxic response to allogenic tumor cells [81,82].

Excess nutrient consumption as well as deficiencies can impair immunity. Numerous studies have linked overnutrition, mainly in the form of fats, with increased cancer incidence and mortality [15]. Even after controlling for calories, it appears that fats exert a strong effect [83,84]. The mechanism by which dietary fats enhance tumorigenesis is still only hypothetical [6,85]. A defect in immune surveillance certainly may play a role here. Obese animals appear to have an increased susceptibility to infection [86]. Among surgical patients, obese individuals suffered from more wound infections than normal weight patients (18.1% vs 7.1%) [87]. Morbidly obese subjects have also been observed to have impaired polymorphonuclear neutrophil (PMN) function [88]. Following intestinal shunt surgery to correct morbid obesity, some aspects of the patients' PMN activity normalized compared to pre-operation depressed values [89]. Obesity is frequently complicated by hyperlipidemia which also may influence immune response [90]. It is hypothesized that increased serum cholesterol can alter cell membrane fluidity, which in turn may affect the functioning of immune cells [91].

Problems with obesity may be compounded by concomitant nutritional deficiencies. In a study of obese children, immune response improved after treating the subclinical iron and zinc deficiencies found in the obese group [92].

In the majority of animal studies, high polyunsaturated fatty acid [PUFA] diets are found to be both immunosupressors and promotors of chemically induced tumorigenesis [93-95]. Diets high in PUFA inhibit T-cell response to mitogens [96,97]. In vitro NK activity appears to be affected by PUFA [98]. A plausible mechanism for the action of PUFA in depressing immunity lies in the biochemical pathway producing series 2 prostaglandins from arachidonic acid. PGE 2, whose synthesis is affected by dietary fat [99], plays a role in depressing immunity. Immunosuppressive PGE is produced in large quantities by tumor cells [100]. Some human studies indicate that PGE production occurs concomitantly with early stages of breast cancer and increases with metastasis of the disease [101,102]. Numerous investigations indicate that PGE 2 can inhibit NK cell activity or cytotoxicity [103,104].

It appears that if the diet contains a minimal level of linoleic acid, the total amount of unsaturation is not as critical as the amount of total fat [105]. Using a chemically induced tumor model, rats fed a high saturated fat diet (containing only 3% linoleate) developed the same number of tumors as those fed a high PUFA (20% sunflower oil) diet [106]. Thus, there appears to be a threshold response such that once a level of about 4% linoleic acid is consumed, total fat intake becomes the key factor [107,108]. It is suggested that a similar pattern of response may occur in humans [109].

Other environmental exposures including smoking and alcohol intake also appear to exert immunosuppressive responses. Heavy cigarette smokers have greatly depressed NK cell activity [110].

A high incidence of a variety of infections in alcoholics has been well documented [111]. This is suggestive of poor immune defense mechanisms [112,113]. Alcohol appears to negatively affect cell mediated immunity including decreased T-cell number, proliferation and migration [114], and NK cell activity. These features are particularly marked with severe hepatocellular injury and/or malnutrition [115].

It is unclear whether the multiple nutritional deficiences that often accompany excessive alcohol consumption affect tumorigenesis directly or indirectly. Deficiencies of vitamin B1, folate, magnesium, iron, zinc, beta carotene [116-118] appear to compound the influence of alcohol on immunity. Lymphopenia is not evident in alcoholic subjects with proper nutrition [119-121]. It is important to note that alcohol consumption in blacks is more strongly associated with malnutrition than it is among whites [122].

Hormonal Effects

Both specific dietary fats and adiposity may influence hormonal factors and that in turn, may affect the risk of cancer in endocrine target tissues. It is known, for example, that specific fatty acids may contribute to peripheral tissue metabolism of estrogens [123]. Obese women have both a greater store of androstenedione and greatly increased efficiency in the conversion of androstenedione to estrone [124]. It is also known that obese individuals have decreased levels of sex hormone binding globulin (SHBG) and concomitantly greater concentrations of unbound steroid hormone levels [125]. Dietary fatty acids have also been shown to inversely affect levels of SHBG [126].

Prostate cancer, whose average annual incidence rate exceeds that of any other cancer site for males, is about twice as high for U.S. blacks as whites [127]. It is widely thought that prostate cancer is determined by factors related to steroid hormone metabolism and nutrition [128]. In a study conducted in Los Angeles it was found that young black men had higher total and free testosterone levels than otherwise comparable white men [129]. Though no direct evidence was available to test the hypothesis, it was proposed that an underlying nutritional difference, possibly related to fat intake, was responsible for these findings. Such a nutritional factor could also explain the striking differences in prostate cancer rates between U.S. and African blacks.

Other Mechanisms:

Besides hormonal and immunological effects, nutritional factors can exert their influence in a number of other ways. It is known, for example, that vitamins C and E, the carotenoids, and selenium greatly influence the probability of lipid peroxidation [130]. Lipid peroxides, in turn, affect cell membrane permeability which may influence carcinogenesis through a number of other mechanisms. Contaminants in food, especially those soluble in lipid, may also be a factor [131]. Again, aggregate fat exposure would mask not only the influence of particular fats but would certainly not allow distinguishing effects of contaminants in fat. It may ultimately rely on measurement of components in tissues, such as adipose tissue.

The interaction of various nutritional factors is illustrated by the effect of nutritional factors on activation of the mixed function oxidases. It is known that alcohol and dietary fat induce these enzymes to different extents in various organs [132]. Substrates for these enzymes are often non-mutagenic/carcinogenic compounds which are metabolized to carcinogenic forms. For example, under the influence of various polyunsaturated fatty acids which mediate prostaglandin endoperoxide synthetase activity in lung tissue, benzo[a]pyrene is converted much more readily to its carcinogenic metabolic product [133] than it otherwise would be. A complete discussion of possible mechanisms of action for these nutritional factors is beyond the scope of this paper. It should be stressed that non-nutritional factors, such as smoking behavior, which appear to differ between whites and blacks, may interact with nutritional factors, whether or not they differ drastically between the races.

Future Studies.

In previous work we discussed various impediments to detecting true diet/cancer relationships [1]. The first of these is the inadequacy of dietary assessment methods to estimate true nutritional intake with sufficient precision, especially over long periods of time. Second is the use of retrospective study designs which require recall of past diet by persons who have cancer. Third is the selection of study groups who typically lack sufficient within-group variability for the dietary factors of interest.

We have also shown that attributable or relative risk differences according to specific risk factors exerted a relatively stronger influence on power to detect an effect than incrementally equivalent increases in incidence rates. These facts have implications for conducting studies of cancer in black populations. Use of dietary assessment methods presents serious problems in terms of providing precise and accurate nutrient data [1]. In previous work, Dr. Willett and colleagues [134] demonstrated the importance of maximizing the accuracy and precision of questionnaire-derived dietary data by fine-tuning the research instruments to the population of interest. Still, even under the best of circumstances these are imprecise tools. Another possibility for overcoming these problems would be to use biological samples as indicators of long-term nutritional exposure. Despite the promise such biochemical markers have for future research, very little work has been done to validate markers against nutritional exposure [1]. This area will probably be of utmost importance as a preliminary step in describing the diet/cancer link.

Although it is known that adipose tissue at various sites and with varying fatty acid composition functions in metabolically distinct manners [124,135], there has been very little work done on describing differences in adiposity in epidemiologic studies. Since blacks are known to have increased peripheral fat deposition compared to whites it would be well-advised to measure differences in within-person fat patterns in future studies. Perhaps some of the rate differences in endocrine-related cancers could be explained by more thorough description of adiposity. Adipose tissue, itself, is also a potentially rich source of long-term (over 2-year) fat and fat-soluble vitamin exposure information [136,137].

Because of the enormous public interest in diet/cancer relationships there is a strong possibility that case/control studies will suffer the irremediable problem of recall bias as well as the perennial problem of imprecision inherent in recalling dietary exposures from the distant past. For this reason, a strong argument could be made against using the case-control design to study diet/cancer relationships.

Despite the fact that many dose-response relationships are not linear, researchers usually assume linearity of effect in selecting populations for study, deciding which variables to measure, and, ultimately, in stipulating models for analysis. As an example of a possible pitfall in making such assumptions, consider the role of dietary fat in breast carcinogenesis. Some animal experiments indicate a maximum effect at about 20-30% of calories, provided a small proportion of calories come from linoleic acid [138]. Small variations in dietary fat may be associated with large variations in other relevant factors, such as bacterial flora [139,140] which, in turn, could exert a powerful effect on steroid turnover in the colon and therefore would affect steroid hormone metabolism in general. Most epidemiologic evidence implicating dietary fat in breast cancer includes a number of women or whole populations who have very low intakes of dietary fat. Because of skewing of fat intake distributions, relationships in ecological data that appear to be modeled adequately using assumptions of linearity may, in fact, be truly curvilinear. Null fat/breast cancer relationships observed in studies conducted using typical Western populations [141] may result from examining a "flat" portion of the response curve.

Study groups should be selected to maximize the probability of detecting true diet/cancer relationships and in contributing to our knowledge about the full range of dose-response relationships. A shortcoming in available data which we noted earlier is that U.S. mortality rates are not disaggregated by race further than non-white versus white. Since blacks comprise the majority of non-whites, the rates reflect mainly black mortality. The other groups that comprise non-whites, however, tend to have lower mortality for many of the sites than do blacks. The real rate differences for blacks are probably even higher than the figures that we gave would indicate.

Since most of the racial differences in rates are probably related to exposures rather than genetics, we should focus more on exposure issues than on group identification per se, perhaps focusing on black subgroups with extreme dietary exposures. Because there may be genetic/environment interactions, it would also be advisable to collect information on family histories of cancer.

In general, overall cancer rates for non-whites, especially males, are increasing faster than those of whites. The most striking differences are among cancer sites most plausibly related to diet. There is no single nutritional or other factor which could explain any large cancer site race-specific rate difference or change in rate over time in a simple, linear fashion. If dietary constituents are truly important in cancer etiology we will need to consider how nutrients interact with one another as well as with other risk factors. This will require great care in selecting populations for study, measuring the factors of interest with precision and minimal bias, and modeling relationships as they truly exist.

Dietary Recommendations

Despite the uncertainty about specific diet/cancer relationships, we are called upon to give dietary advice. As with the hygienic movement of the nineteenth and early twentieth centuries, we can decide on reasonable recommendations based on limited data and without complete knowledge of specific mechanisms of action. In general, we support the dietary goals for the United States [142] which are aimed at all major sources of chronic disease mortality in the U.S. population. The American Health Foundation Food Plan recommends a further reduction in total fat to 25% of total calories, a P:S ratio of 0.7, cholesterol reduction to 200 mg/day, and an increase in dietary fiber to 30g/day.

Acknowledgements

This work was supported by National Cancer Institute Program Project Grant No. CA32617 and Center Grant No. CA17613, and American Cancer Society Special Institutional Gant SIG-8.

We acknowldege Verna Harrison for manuscript preparation, Roslyn Fieland for preparing tabular and illustrative material, and John Barbato for photographing the illustrations.

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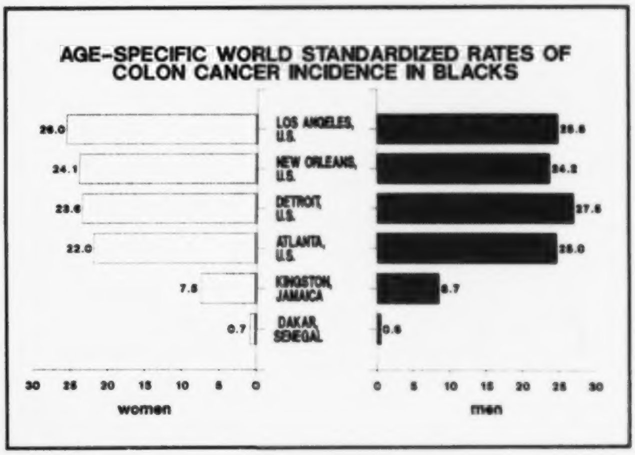


Figure 1

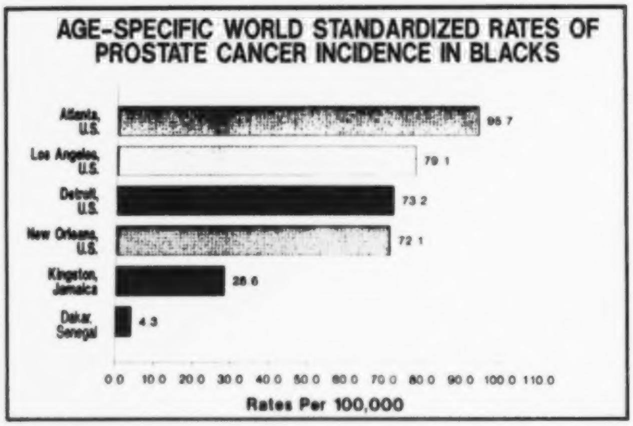


Figure 3

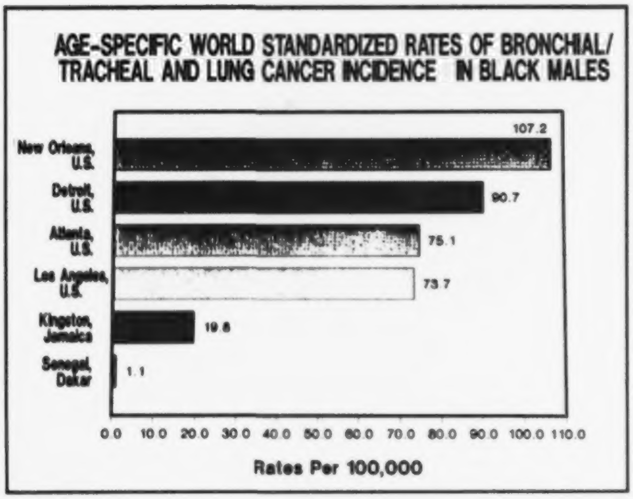


Figure 2

Figures 1, 2, and 3—Data are from : Waterhouse J., Muir C., Correa P., Powell J.: Cancer Incidence on Five Continents - vol. 3 Lyon: International Agency for Research on Cancer, 1976. reference = [2]

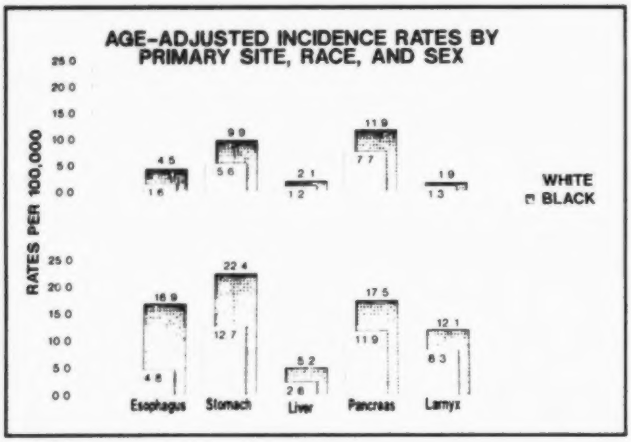


Figure 4

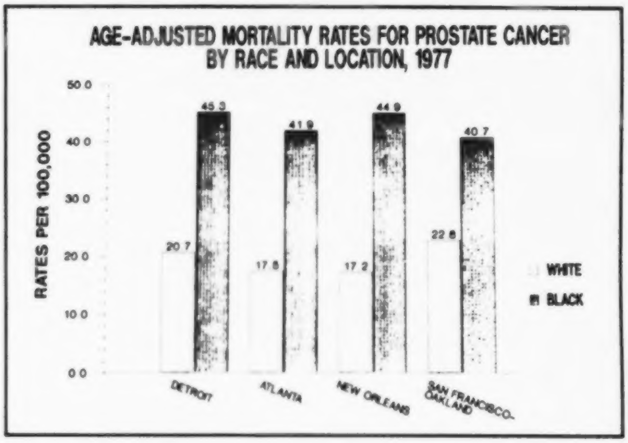
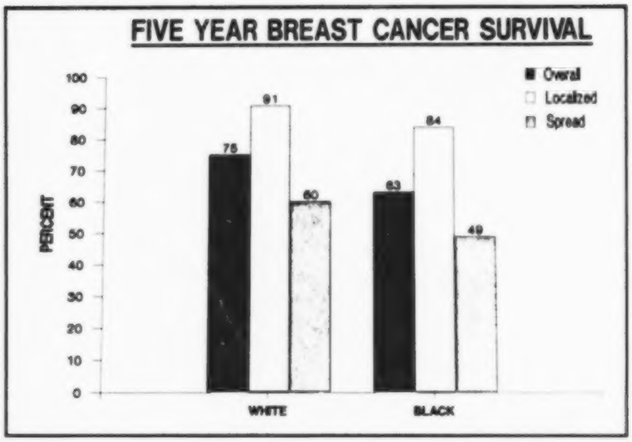


Figure 5

Figures 4 and 5 - Data are from: National Cancer Institute: Surveillance, Epidemiology and End Results: Incidence and Mortality Data, 1973-77. (NIH publ No. 81-2330) NCI Monograph No. 57. Bethesda, MD: NCI June 1981. reference= [153]



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Data are from: **Cancer Statistics, 1987** CA-A Cancer J. 37: 1987. reference = [154]

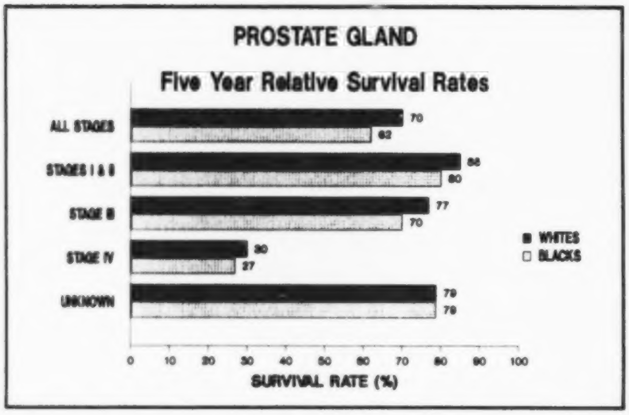

Figure 7

Data are from: National Cancer Institute. **Cancer Among Blacks and Other Minorities: Statistical Profiles.** Washington, D.C.: U.S. Department of Health and Human Services, 1986 reference = [155]

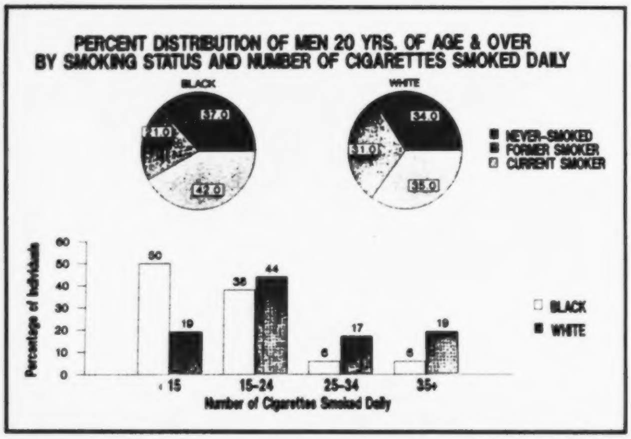

Figure 8—Data are from: Schoenborn CA, Cohen BH; Trends in smoking, alcohol, and other health practices among U.S. adults: United States, 1983. Advance data No. 118[suppl] DHHS Publ. No. [PHS] 86-1250 Hyattsville, MD: National Center for Health Statistics, June 30, 1986. reference = [156]

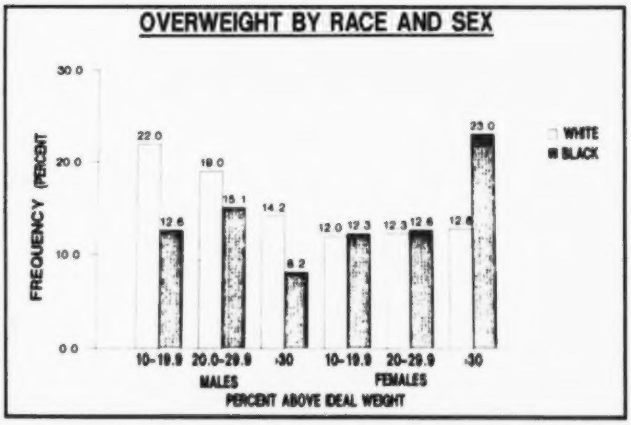

Figure 9: Data are from: National Center for Health Statistics: Provisional Data from the Health Promotion and Disease Prevention Supplement to the National Health Interview Survey; United States, Jan-Mar, 1985. Advancedata No. 113 DHHS Publ. No. [PHS]86-1250 Hyattsville, MD: National Center for Health Statistics, Nov. 15, 1985. reference = [157]

Nutrition And Immunity: Discussion

Enwonwu, CO.

My question is for Robert Suskind. The traditional idea that kwashiorkor is due entirely to protein deficiency occurring concomitantly with adequate or excessive caloric intake is now seriously questioned, and perhaps requires some revision. There is currently good suggestive evidence for an important role for free radical damage in the genesis of kwashiorkor as opposed to marasmus. Indeed, it is possible that such mycotoxins as aflatoxin B1 precipitate clinical kwashiorkor in the protein deprived child by generating excessive amounts of free radicals in the presence of inadequate amounts of antioxidants like vitamin E, zinc, selenium, glutathione and others. Would you care to comment on this please?

Suskind, RM.

Obviously, there are a number of factors involved, and I don't think kwashiorkor is necessarily a question of dietary intake. I think it is probably more a manifestation of the role of infections. In marasmus, caloric intake is basically inadequate, and protein intake is probably semi-deficient. As a result, the child stops growing. My own perception of the development of kwashiorkor is entirely different. You can have a normally nourished child develop kwashiorkor merely as a result of infection, trauma or some stress which elicits a catabolic response. The resulting exaggerated loss of nitrogen is probably more important than the inadequate protein intake. This is the way I perceive the development of marasmus and kwashiorkor. Obviously, there are other factors.

Adebonojo, FO.

I used to tell my students in Africa that marasmus was usually found in children aged 6-12 months. Although some children older than 1 year develop marasmus, the predominant age group where I worked in Africa was below 1 year, whilst kwashiorkor was usually seen in much older children - certainly after one year of age. I am just wondering whether you have used the concept of age of onset of malnutrition to separate kwashiorkor from marasmus.

Suskind, RM.

The observations in Africa which you have raised are valid in terms of children under 1 year of age developing marasmus, and kwashiorkor being found mainly at 2-3 years of age. In this country you may find some children who develop the marasmic type of picture as a result of inadequate calorie intake relative to their needs. You can have children with cancer, chronic renal disease, cystic fibrosis, etc who have a relative calorie deficit, and manifest this in failure to thrive. I think that the mechanism is more important rather than the age, and therefore what you saw in children who developed kwashiorkor was probably more a manifestation of susceptibility to infection. Really, I have never seen a child develop kwashiorkor who has not had a preceeding episode of infection, e.g. chronic diarrhea, with resultant increased nitrogen loss.

Adebonojo, FO.

Will the immunologic responses of these children distinguish those with kwashiorkor from those with marasmus?

Suskind, RM.

The data I showed you this morning indicated that children with kwashiorkor did have significantly lower levels of complement factors than those with marasmus. In terms of lymphocyte function, we did not see any difference between marasmic and kwashiorkor children. Data from individuals in this country with anorexia nervosa, which is really marasmus of teenagers, show intact immune system. Patients with anorexia nervosa usually take vitamin and mineral supplements, and therefore may have only protein and calorie deprivation. In the populations with marasmus and kwashiorkor, there are multinutrient interactions with the immune system, and it is possible that it is not the protein - calorie deprivation but rather deprivation of the micronutrients that may be affecting the immune system. The latter conclusion is supported by data from the patients with anorexia nervosa.

Enwonwu, CO.

This is a very important subject especially in view of its world-wide implications. I am not in any way trying to minimize the importance of the excellent contributions of Dr. Cicely Williams on the genesis of kwashiorkor. Very early studies in this area focussed mainly on the role of the major food nutrients - particularly proteins and carbohydrates. There was very little known then about the potential role of the micronutrients, particularly the essential minerals. There is now increasing evidence that such minerals as zinc, selenium, and others play important roles in the genesis of kwashiorkor. As a matter of fact, recent studies by Golden and his colleagues have demonstrated clear cut differences between kwashiorkor and marasmus with special reference to blood levels of selenium and glutathione. Also recently, there are suggestions from the Liverpool University group headed by Professor Hendrickse that aflatoxin ingestion from contaminated foods may be important in the genesis of kwashiorkor. A unitary hypothesis that seems to accommodate all the reported causes of kwashiorkor seems to revolve around free radical pathology and the important role of some micronutrients as free radical scavengers. Vitamin E, B-carotene, zinc, ascorbic acid, and glutathione are among the important free radical scavengers, and most of them are depleted in the child with kwashiorkor. Also, aflatoxin may act by generating free radicals. We now know, for example, that serum albumin level has very little meaningful relationship to the degree of edema in kwashiorkor, and there is no convincing evidence for a cause - effect relationship between reduced serum albumin and the edema of kwashiorkor.

Suskind, RM.

In terms of the development of edema, there is multifactorial causation as you mentioned. It could be a result of the effects of malnutrition on the capillary basement membrane in terms of its permeability. I think the points you have made with regard to zinc and selenium are important because the children with kwashiorkor do have deficiencies of these micronutrients. All I was relating in terms of development of kwashiorkor was the pathogenesis of decrease in serum albumin. In my own thinking, the basic reason for decrease in serum albumin is reduction of available amino acids for synthesis of this serum protein. The available amino acids appear to be preferentially used for other functions such as synthesis of acute phase reactants.

Margen, Sheldon.

I have seen a child go from marasmus to kwashiorkor in 48 hours. In most of these children, one is dealing with interactions between infections, malnutrition, immune dysfunction and other adverse environmental factors.

Suskind, RM.

The interaction between malnutrition and infection is obviously a very important interaction, and very complex. It involves also the immune system. So far the details have been carefully dissected out only in the animal models of malnutrition. We have shown in the latter, the severe effects of deficiencies of vitamin A, iron, and zinc on the immune system.

Byron, JW.
Severe bone marrow depression is a major consequence of treating AIDS patients with the well known AZT. This can be potentiated by the use of some other drugs like PCP, antifolates, and others. What is the present awareness of these problems in terms of preparing AIDS patients about the complications of these drugs - especially in terms of getting them nutritionally prepared for drug therapy?

Collins, Candy.
Most of our patients on AZT are prepared nutritionally. Adequate food intake is stressed.

Suskind, RM.
AZT in AIDS patients suppresses the bone marrow, and this can be compounded if the patients are malnourished. Hence the need to accentuate nutritional support in these patients.

Carter, JP.
My first question is for Ms. Collins. In a patient with AIDS - related complex, constitutional symptoms, has lost his appetite but has no opportunistic infections, - what can you do to overcome his anorexia and put him in a positive nitrogen balance?

Collins, C.
I usually see them at the ARC stage, in a clinic setting. I usually review their diets with them to make sure they have enough calories and proteins, and where necessary, supplements of micronutrients are recommended.

Carter, JP.
What kind of success do you have?

Collins, Candy.
Our aim is to get them to eat well and maintain their body weight or at least, prevent further weight loss.

Carter, JP.
The best you really can do is to help them maintain their body weight rather than regain weight already lost. If you can't reverse the weight loss in the ARC stage, it will be at least ten times as difficult once they develop opportunistic infections like pneumocystis carinii pneumonia.
The other question I have is for Dr. Hebert. I am intrigued by the possible connection between menthol cigarette smoking and esophageal cancer. Do you have any idea of the mechanisms involved? Are nitrosamines involved?

Herbert, JR.
There are several possibilities, namely (i) Direct effects of menthol and its products of combustion; An article published in Nature in 1965 addressed the issue of what happens when menthol is combusted. All sorts of interesting compounds are byproducts of the combustion. (ii) Menthol heightens the sensation to cold but attenuates sensation to heat, in mild aequous solution that simulates conditions in the mouth - the latter property obviously permits ingestion of very hot beverages without much discomfort.

Grossman, EM.
I am a bit skeptical about some of the data derived from population studies on fat/colon cancer relationship. The picture is obviously not clearcut. Experimental animal studies indicate that the type of fat is important. We don't know how the interaction between fiber and fat fits into the picture. There is also evidence that it may not be fat per se but total calorie intake that is the culprit.

Herbert.
Obviously, there are lots of questions involved. It is not easy to disentangle all the interactions in the human. Individual fats may be contributing to the problem, but total amount is just as important. Ecological studies do have a lot of problems but certainly, the strongest relationship points to dietary fat.

Anonymous.

My question is for Ms. Collins, Is there really anything that can be done from the nutritional point of view to protect children of AIDS mothers, or at least promote their resistance?

Collins, Candy,

Nutritional support is extremely important for the children.

Suskind, RM.

I will go further to recommend that these be put on 150 percent of the RDA.

Adebonojo, FO.

Will you do that even if they are growing normally?

Suskind, RM.

You monitor their growth. The studies have not been systematically done yet, but I would guess that vigorous, preventive nutritional support may be helpful.

Anonymous.

Mention was made by Dr. Hebert that height may be one of the risk factors in cancer causation. At what height is this of importance?

Herbert JR.

About 7 feet 6 inches - laughter. In all seriousness, the strongest relationship I have read for height and cancer is a publication on endometrial cancer by Dr. Wynder and his colleagues. A similar relationship between weight, and endometrial cancer is also speculated.

FIFTH SCIENTIFIC SESSION

Sickle Cell Anemia

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Sickle Cell Anemia Overview

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Manuscript Not Available

Nutritional Costs of Sickle Cell Anemia

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Introduction

Sickle cell disease (HbSS) or sickle cell anemia, is an autosomal recessive condition in which glutamic acid in position 6 of the B-chain of the hemoglobin molecule is replaced by valine. HbSS affects many nationalities, and is the major genetic disease found in blacks. It is characterized by painful crises of variable intensity and frequency, hemolytic anemia resulting from accelerated breakdown of the red blood cells (RBC), defective immune function, marked functional impairment in various organs as a consequence of repeated vaso-occlusive insults (1-4). Another common feature of the victim of HbSS is growth impairment (1, 5, 6). The latter is not due to any defect in response of pituitary growth hormone to growth releasing factor (7), but rather to inadequate nutrition and repeated infections (1). The importance of malnutrition and other environmental factors in the clinical severity of HbSS is exemplified by the reported observations to the effect that in Africa, unlike in the USA, this genetic condition is generally not compatible with a prolonged, trouble-free survival (3). Konotey-Ahulu (8), who has carried out extensive investigation of the disease in West Africa, has confirmed that a raised standard of living including proper nutrition, gives the victim of HbSS a longer useful life.

Jackson (9, 10) considers HbSS an ideal model of an elevated persistent drain on the nutrient economy of the body. Two recent comprehensive reviews have examined the requirements for essential nutrients and energy in HbSS (11, 12). Because of the constraint of time, my presentation this morning will focus primarily on nitrogen metabolism in HbSS. Subsequent speakers will address specifically, the requirements for energy and the essential micronutrients (vitamins and mineral elements) in this disease, which so far, has no known cure.

Pathophysiological features of HbSS: Brief Review

The life span of normal RBC varies from about 90 days in children to 120 days in adults. A healthy 70-kg adult man has about 750-900 g of hemoglobin of which approximately 0.3 g is destroyed or synthesized every hour (4, 13). This is equivalent to a daily turnover of about 7.2 g of hemoglobin. In HbSS, rates of destruction of the RBC may be as high as 6- to 20- fold normal (1, 4, 14, 15). Serjeant (1) has estimated a minimum of at least sixfold increase in effective hemoglobin synthetic rate from the normal 6 - 7 g/day to no less than 40 g/day in HbSS. At this level of hemoglobin production, the bone marrow reaches the limit of its ability to compensate for accelerated loss of RBC (4). The compensation index (ratio of production rate to destruction rate of RBC) in HbSS patients with hemoglobin levels in the range of 7.0 to 8.8 g/100 mL, is reported to vary from 0.53 to 0.70 (13). The latter implies an hemoglobin destruction rate of the order of 57.0 to 75.5 g/d if the synthetic rate is assumed to be 40 g/d.

In HbSS, about 33 percent of the hemolysis occurs intravascularly and plasma hemoglobin levels may be as high as 10-25 mg/100 ml (4). It is estimated that the amount of hemoglobin required to maintain a plasma level of this range is at least 10.8 g/d or 1.5 - fold normal daily production of hemoglobin (4, 15). Destruction of lg of hemoglobin releases 35 mg of bilirubin, and in a healthy 70kg man, this amounts to a daily production of no less than 220 mg of bilirubin. In HbSS, daily production of bilirubin may be as high as 2000 - 2,600 mg, resulting in a prominent increase in fecal nitrogen loss since most of the bilirubin produced in the body is an end product of metabolism (9, 16, 17).

Evaluation of nutrient losses and/or requirements in HbSS must of necessity take into consideration the functional changes in such organs as the kidney (1, 18). These changes usually result from the hemodynamic alterations of chronic anemia, and as consequences of repeated vaso-occlusion. Impaired renal - concentrating ability is a common and early feature of HbSS, and hematuria is the most dramatic of the renal manifestations of the disease (18). Proteinuria is found in 29-50 percent of HbSS victims aged 10 years and older (1), and those who survive to adult life develope progressive renal failure at an early age (10, 16).

Nitrogen - Energy Metabolism in Sickle Cell Anemia.

Maintenance of the circulating red cell mass in HbSS implies a profoundly increased demand for protein synthesis, and it is well known that formation of the peptide linkage is one of the most expensive intracellular processes in terms of energy utilization. My colleague, Dr. Adebonojo, will discuss energy need in HbSS in greater depth. I do however wish to point out that synthesis of each peptide bond requires an energy input equivalent to at least 5 to 6 moles of adenosine triphosphate [ATP] (19), a value synonymous with about 100-120 kcal/bond in terms of food energy (20). Hunt (21) estimates that polymerization of each of the four globin chains of the hemoglobin molecule requires about 600 ATP to adenosine diphosphate hydrolysis equivalents. Additional energy is required for provision of the tertiary structure of proteins, synthesis of the multiprotein complexes in the RBC membrane, breakdown of proteins with release of the constituent amino acids into the free pool, and the active transport of materials across the cell membranes. Even the formation and excretion of waste products require energy, and it has for example been shown that on a daily dietary intake of 200 mg N/kg body weight, the adult HbSS patient produces about 238 mg of urea N/kg compared with only 139 mg N/kg by the healthy individual (10). It is therefore not surprising that energy deficit is probably the most limiting nutritional factor in the HbSS (9, 22).

The resting metabolic rate increases by about 20 percent in HbSS (from 96kJ/kg/d in healthy persons to 118 kJ/kg/d in HbSS patients), and it is estimated that as much as 48 percent of the total energy expenditure is consumed by activities related to protein turnover, compared with 36 percent in the healthy individual (9). Jackson (9, 10, 16), who has carried out comprehensive studies of nitrogen (N) metabolism in HbSS, reports that on a dietary protein intake of 1.0 to 1.5 g/kg/d, protein turnover in the HbSS, victim relative to findings in healthy individuals, increases by about 63 percent, representing elevation in both synthesis and degradation. Although turnover of proteins in the body is associated with extensive reutilization of amino acids for synthesis of new proteins, the latter process is by no means completely efficient as evidenced by the loss of some amino acids through oxidative catabolism and by excretion (23). Thus, the victim of HbSS usually requires higher than normal dietary N intake to stay in positive balance. This is not due to poor intestinal digestion and absorption but rather to inefficient utilization of ingested protein resulting in a profound increase in urinary and fecal losses of nitrogen (9, 10, 22, 24).

Requirements For Specific Amino Acids in HbSS.

The increased requirement for N in HbSS does not necessarily imply that all the amino acids are needed in increased amounts. It is also possible that in this genetic disorder, certain dietary dispensable amino acids found in high concentrations in the hemoglobin molecule, may become conditionally essential. An analogous situation is the severely burned patient where the dietary nonessential amino acid proline, a prominent constituent of collagen, becomes conditionally essential (23). Hemoglobin is a structurally unbalanced protein, totally devoid of isoleucine but very rich in valine and leucine which together make up about 20 percent of the total amino acid residues. This protein also has an excess of glycine, and contains about 8 per cent histidine.

Synthesis of each heme ring of hemoglobin requires 8 molecules of glycine, suggesting that the prominent irreversible fecal loss of the end product of heme metabolism, bilirubin, in HbSS would accentuate the demand for glycine. Jackson (10, 16) estimates a daily loss of 2-4g of glycine through this route in HbSS. The high demand for this amino acid, which is utilized to form positions 4, 5 and 7 of the purine skeleton, is further underscored by the increased production and loss of uric acid in HbSS (1, 25). Recent findings of increased urinary excretion of 5-oxoproline (pyroglutamic aciduria) in HbSS strengthens the belief that victims of this disease may exist in a chronically precarious state with respect to glycine sufficiency (26). The latter is consistent with the reported observation in HbSS of reduced RBC content of glutathione (GSH), an important free radical scavenger (16).

Additional to glycine, other amino acids which may require special attention in HbSS are taurine and histidine. The increased loss of taurine-conjugated bile salts in the feces in HbSS constitutes a drain on the pool of sulphur amino acids. Even in the healthy individual, the endogenous renal clearance rates of histidine, glycine and taurine are very high relative to the rates for the other amino acids (27). Urine/plasma ratios for various amino acids in the healthy human adult, as reported by Evered (28), are histidine (6.6), taurine (5.5), glycine (4.0), tyrosine (1.8), methionine (1.2), phenylalanine (1.2), isoleucine (1.1), leucine (0.4), lysine (0.3), and valine (0.2), thus confirming the relatively inefficient renal reabsorption of the filtered load of histidine, glycine, and the non-protein amino acid, taurine. A more recent report (29) indicates much higher fractional excretion (FE) of taurine, as well as of 1-methylhistidine and 3-methylhistidine, in the normal human, than previously reported. The principal determinants of the FE of an amino acid are the plasma concentration of the latter, the glomerular filtration rate (GFR), and the characteristics of renal tubular reabsorption. Excretion rates of most amino acids increase 2- to 10-fold above normal in chronic renal failure (30), and the latter is not an uncommon finding in victims of HbSS who survive to adult life (10, 16, 18).

Of particular interest to our research effort on the dietary management of HbSS patients, is the impact of accelerated protein breakdown usually associated with this genetic disease, on histidine metabolism as well as its loss in urine. This amino acid is now considered a dietary indespensable amino acid not only for children but also for human adults, especially those with chronic diseases (31). Histidine is required for maintenance of N balance and mitochondrial membrane integrity, as well as for normal erythropoiesis (31-33). Activity of erythrocyte d-aminolevulinate dehydratase (porphobilinogen , EC4.2.1.24), a key enzyme in heme biosynthesis, is prominently reduced in histidine depletion (34). The daily requirements for this amino acid vary from 33 mg/kg body weight in infants to about 8-10 mg/kg in adults (35).

Based on the assumption of a normal dietary histidine content of 3g/100g protein, Silbernagl (29) has calculated the FE of histidine in humans (about 6 percent of the filtered load), to approximate 5 percent of the daily intake of the amino acid. Although the amino acid resorptive capacity of the renal tubules may not be exhausted by as much as 1.5 - fold rise in the filtered load, it is now believed that, for some amino acids particularly histidine and its metabolic products 1-

and 3- methylhistidines, a significant increase in fractional excretion (FE) may result from increased filtered load (29). It is in this light that one must evaluate the potential consequences of the extensive protein turnover found in HbSS. The concentration of histidine ranks third among eight indispensable amino acids in the skeletal muscle, which contains no less than 50 percent of the total pool of each amino acid in the body (36). Even the daily destruction in HbSS of 57-75g of hemoglobin, with its high content of histidine (about 8 percent), releases no less than 4.6-6.0g of free histidine into the circulation every day (11). The resulting changes in plasma concentration of histidine will impact on the FE of the amino acid through an increase in the GFR.

Recent reports of limited glycine availability in HbSS (26) lend further support to the possibilities of impaired histidine metabolism in this hereditary disorder. The major degradative route of histidine in the liver is deamination catalyzed by histidase (L-histidine ammonia-lyase, EC 4.3.1.3) to yield urocanic acid, leading to production of formiminoglutamic acid (FIGLU) which is then converted to glutamate and formiminotetrahydrofolate (TH4). The latter enters the one-carbon pool, and free TH4 must be regenerated to promote further catabolism of histidine. Regeneration of free tetrahydrofolate utilizes pathways involving formation of serine from methylene - TH4 and glycine, and formation of methionine from methyl-TH4 and (37).

Status of Essential Minerals and Vitamins.

Physiological requirements for minerals and vitamins are usually closely related to the needs for calories and nitrogen. It is not therefore surprising that the chronic stress of HbSS increases the requirements for most of the essential micronutrients (11). Potassium balance for example, is very closely related to N balance. Since proteins, particularly the enzymes, hold and lock many essential minerals like zinc, selenium, copper, magnesium and manganese in the tissues (38), it is to be expected that HbSS, with its profound negative effect on nitrogen metabolism, will elicit losses of many of these minerals and thus increased dietary requirements for them.

Selenium status is significantly compromised in the HbSS patient (39). The selenoenzyme, glutathione peroxidase (GSHpx) accounts for 30-40 percent of the total body content of this essential trace element in mammals (40), and this enzyme, through action on hydroperoxides of unsaturated fatty acids, prevents structural damage and disruption of cellular integrity caused by peroxidation of membranes. There is some controversy regarding the status of GSHpx activity in HbSS, with Chiu and Lubin (41) reporting a marked increase while other investigators have observed a significant decrease (42). The latter view is consistent with Golden's finding (38) of low GSHpx activity in selenium deficiency.

Perhaps, the essential mineral element which has received the greatest research attention in HbSS in recent years is zinc. It is claimed that several clinical features commonly associated with HbSS, return to normal following dietary zinc supplementation (43, 44), and that the significantly reduced zinc level in plasma, neutrophils, red blood cells, and hair in adult HbSS is due mainly to increased urinary loss of the essential trace element (44). The hyperzincuria in HbSS increases with age, and in adult patients, may be as high as 4-5 fold normal values (43). Our laboratory is exploring the possibility that zinc depletion in HbSS is preferentially linked to distorted histidine metabolism.

Close metabolic relationships exist between histidine and zinc (45). Functional zinc in metallo-enzymes is associated with histidine residues in protein molecules (46), and there is good evidence from studies in the human (47, 48) and in experimental animals (49, 50) that plasma concentrations of certain amino acids, particularly histidine and cysteine, may be major determinants of urinary zinc excretion. Oral or intravenous administration of histidine elicits a dose-dependent hyperzincuria (48). It is our hypothesis that in HbSS, the profoundly increased turnover of proteins, and particularly of hemoglobin with its high content of histidine, increases the circulating pool of free histidine. This situation promotes urinary losses of the amino acid and zinc. The hyperzincuria further impairs histidine metabolism as evidenced by findings in experimental animals where zinc deficiency promotes a three to fourfold increase in urinary excretion of the amino acid compared to observations in zinc-supplemented animals (45). Additional support for this hypothesis is provided by reported observations that several characteristic features usually associated with HbSS, such as anorexia, defective erythropoiesis, malaise, changes in emotional status, and a skin rash, have been variously described in deficiences of zinc (44) and histidine (31, 33, 34).

Other mineral elements which have been studied in HbSS include calcium and iron. Calcium accumulates above normal levels in the RBCs in HbSS, and may be an important factor in the clinical severity of the disease (3).
In many patients with HbSS, and particularly in those who have received multiple blood transfusions, sequestration of iron in the hepatic parenchymal cells is a common finding. This internal, cellular "free" iron pool has the disadvantage of effectively promoting the formation of damaging hydroxyl radical (51). Nevertheless, studies of twenty-five Asiatic Indian children with HbSS, most of whom had never received blood transfusion, showed widespread severe iron deficiency (52). Similarly, high iron excretion (0.5 mg or more daily) has also been reported in urine samples from 27 of 31 adult HbSS patients (53), an observation consistent with the marked intravascular hemolysis of RBC and the impaired renal function usually associated with this disorder especially in adult life.

With regard to the vitamins, increased dietary requirement for folate is a common finding in conditions of greatly reduced RBC lifespan (54), and episodes of folic acid deficiency as well as megaloblastic erythropoiesis have been noted in HbSS despite what appears to be adequate intake of the vitamin (55). Serjeant (1) estimates that reversal of the megaloblastic changes in HbSS may at times require the ingestion of as much as tenfold the normal daily needs of the vitamin. It is possible that folate deficiency in HbSS may be due in part to poor intestinal absorption in addition to increased utilization of the vitamin by the bone marrow. Polyglutamyl folate, the predominant form of the vitamin in foods, is hydrolyzed in the human intestine by the enzyme pteroylpolyglutamate hydrolase (PPH) found on the luminal brush border membrane. Human PPH is a zinc-dependent exopeptidase whose activity may be impaired by the zinc deficiency in HbSS (44). The folate deficiency in HbSS will impact adversely on the metabolism of histidine (56), and perhaps also, on the glycine cleavage system for endogenous synthesis of glycine (9, 16). Human requirement for riboflavin (vitamin B2) increases with increased energy expenditure (57). It is not therefore surprising that B2 deficiency as reflected by significantly reduced activity of the flavin-dependent enzyme, erythrocyte glutathione reductase (NADPH: glutathione oxidoreductase; EC1.6.4.2) is seen in patients with HbSS (58). The importance of adequate vitamin B2 status in the clinical management of HbSS is underscored by reported in vivo observations of impaired production and reduced survival of RBC in experimentally induced ariboflavinosis in humans and animals (59).

Another water-soluble vitamin that has been reasonably well studied in HbSS is vitamin B6, and significant reduction in plasma pyriodoxal 5'-phosphate (PLP) concentration with concomitant elevation in RBC content of the vitamer, has been observed (60). There are suggestions that adequate PLP status inhibits sickling of RBC in HbSS (61). Poor status of vitamin B6 in HbSS may not necessarily be due to inadequate dietary intake but rather to riboflavin (B2) deficiency as demonstrated by the observation that RBCs from patients with B-thalassemia exhibit reduced in vitro rate of conversion of pyridoxine to the active PLP, which can be corrected if the patients are supplemented with riboflavin (62). Similarly, nicotinic acid is essential for the formation of PLP, and there is also a poorly defined but important interaction between vitamin B6 and ascorbic acid as evidenced by the reversible 3.5-fold increase in free urinary pyridoxne level in vitamin C deficiency (62). Significant reductions in levels of vitamin C in plasma (63) and leukocytes (64) have been noted in HbSS patients compared with normal controls.

With regard to the fat-soluble vitamins, several investigators have demonstrated prominent reductions in blood circulating levels of vitamin A (65), retinol-binding protein (58), and vitamin E (58, 66, 67). Vitamin E deficiency in HbSS may be due to impaired intestinal absorption (41) and/or to increased oxidative destruction of the RBC membrane-bound vitamin (66). Vitamin E is perhaps the most important lipid-soluble antioxidant, and its regeneration from the vitamin E radical state is defective in the absence of adequate vitamin C (68, 69).

Many of the trace elements and vitamins function as important free radical scavengers (68-70), and their relevance to the clinical severity of HbSS will be fully discussed by my colleague, Dr. Stone, who has carried out extensive studies on this subject. I do however wish to point out the important studies of Kay and her colleagues (71) demonstrating that vitamin E deficiency, by eliciting rapid emergence of "senescent cell antigen" on the RBC membrane, causes premature aging of the RBCs with increased IgG binding to these erythrocytes. It would thus appear that in malnourished HbSS patients, the macrophages will recognize the RBCs not only as structurally abnormal but also as prematurely "senescent" (71, 72).

Conclusion.

The stress of HbSS produces caloric inefficiency and a marked increase in resting metabolic rate. Nitrogen metabolism is set at a higher than normal dietary intake because of inefficient utilization of ingested protein, and is adversely affected by the demands for synthesis of an excessive amount of a single protein, hemoglobin (9, 10, 16). Incomplete but compelling data suggest that this genetic disorder may have special amino acid requirements. The needs for most vitamins and essential mineral elements so far studied, are increased in HbSS. Many of these micronutrients, particularly vitamin E, ascorbic acid, B-carotene, zinc, selenium and copper, are important free radical scavengers, and thus play crucial roles in retarding premature aging of the RBCs caused by peroxidation of cellular membranes (68-72). Dietary management of the HbSS victim must also take into account the nutritional impact of frequent bacterial infections (1) as well as the accelerated renal losses of nutrients which may occur as a consequence of repeated vaso-occlusive insults to the kidneys (18).

Acknowledgement

This project was financially supported by the General Foods Fund, Inc.

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Energy Balance In Sickle Cell Disease

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Introduction

The concept of energy balance implies that the equation between the energy introduced into a system (Energy In) must equal the energy expended and lost by the system (Energy Out) in order to maintain a neutral energy state. When the energy in and out of a system are unequal, two possibilities exist. One possibility, a state of positive energy balance, occurs such as in the obese state in which "energy in" exceeds "energy out". Conversely, when "energy out" exceeds "energy in", a state of "negative energy balance" results such as in protein-energy malnutrition and probably the case in sickle cell disease (SCD).

For purposes of this presentation, it is essential to define the various components of "energy in" and "energy out". Under normal circumstances, "energy in" is governed by daily energy intakes in the forms of energy-rich foods and liquids and is dependent upon the digestive and absorptive capacities of the gastrointestinal tract. "Energy out", on the other hand represents a sum of the energy required by the system for survival, normal growth and activities as well as normal and abnormal losses.

With respect to intake, little is known about the true energy requirement in SCD. In the 1982 edition of "Blood and Its Disorders", Hardisty and Weatherall (1) emphasized the degree of paucity of information on this subject. Concerning the growth and development of children with sickle cell disease, they noted that "good data" simply do not exist and further observed that most of the information available is anecdotal and limited. In a 1987 review of the literature, Reed, et (2) noted that "sickle cell disease remains one chronic illness in which the role of nutritional factors has not been thoroughly and systematically examined". With respect to macronutrients, especially the daily requirements for calories and protein, they observed that these have just recently drawn some attention. Even some of the more recent studies (3,4) have been severely flawed by such things as small sample size, lack of appropriate controls and other relevant methodological problems.

Because of their flaws, results of the few studies on the food intake of children with SCD have been contradictory. One investigation (5) reports seemingly normal intakes, others (3,4) report decreased intakes in SCD patients. Several of the factors which were not examined but which could influence intakes, such as the chronicity of the patient's disease, frequency of hospitalization, anorexia and other complications of the disease will be considered later in this presentation.

Studies on the digestive and absorptive functions of the gastrointestinal tract in patients with SCD have revealed largely normal findings (3,6,7). In a few instances when intestinal biopsies have been performed, normal findings have been reported (3,6). Thus, the little available information on food intake and on the digestive and absorptive capacities of patients with SCD appeared to suggest that the problem of negative energy balance in SCD is not due to problems in intake. However, the study by Heymann, et al (3) seems to suggest that there may be a problem with the amount of intake in the patient with SCD relative to his total needs; especially so, since consistent daily nutritional supplementation by nasogastric intubation produces a rapid sustained growth and a strikingly reduced incidence of pain crises and infections in the two children in which it has been used. Like other studies, this is not a properly controlled study and the numbers are too few.

The question remains however: Does the child with SCD have greater requirements for energy than the normal child? If so, which components of energy requirement(s) are increased? This question leads to a consideration of each of the components of energy requirements in children and how they could be affected in sickle cell disease.

Energy Requirements in Children

Basal Metabolic Energy Requirement:

Normally, about 60 to 70 percent of the total energy requirement of a normal child goes to meet basal metabolic needs. This need is the first to be met by the intake. Any increase in the metabolic rate might be expected to increase the energy requirement of the child with SCD. Clearly such an increase occurs in patients with SCD.

The multiple complications of SCD, stressing as they do the entire body system, are bound to produce significant perturbations in the basal energy requirement of the child with SCD. Some of these derangements and their metabolic consequences are explored below.

The usual life span of the normal erythrocyte is 90 days in infants and children and about 120 days in adults. Sickle erythrocyte has a life span of 15 to 20 days, or approximately 15 percent of normal (8 a,b). Additionally, the synthesis of cell membranes and their normal functioning have been estimated to account for as much as 50 to 60 per cent of basal energy needs. Thus, a five to six fold increase in red cell membrane synthesis would be expected to extract significantly increased cost on the basal energy metabolism of the body. A crude guess is that this increase may well be in excess of 20 percent of basal needs. Recent findings of increased endogenous carbon monoxide production in SCD suggest also increased bone marrow activity.(9)

There is a good deal of indirect evidence suggesting that an increased metabolic rate exists in patients with SCD (4,10-13). For example, increased purine metabolism has been reported in patients with SCD, reflected in increased urinary urate excretions (4,10,11) and higher serum urate concentrations (13). Odonkor, et al, (12) have reported negative nitrogen balance, at all levels of nitrogen intakes, in teenage patients with SCD. However, this study failed to control for calorie intake as a function of body size and sickle cell patients were usually lighter in weight than the control subjects. In contrast, the study by Heymann, et al (3) found normal nitrogen balance in two of the three sickle cell patients they studied. When age-matched controls with similar thyroid hormone levels were compared, Odonkor, et al (14) found significantly higher metabolic rate in eight teen age patients with SCD than in the controls. Thus, it would appear that when properly controlled, the basal metabolic rate is higher in patients with SCD than in control subjects.

Patients with SCD experience various types of crises-hemolytic, aplastic, sequestration and vaso-occlusive, painful varieties. These must, quite clearly, stress the metabolic state of the child. There is no information on the energy costs of such crises. However, the study by Heymann, et al (3) in which children with SCD, given significantly increased nutritional supplementation, had significant reduction in frequency of crises episodes, suggest that there may be a relationship between the energy needs of these children and their crises episodes.

Also, infections associated frequently with tissue destruction, repair and febrile reactions, are common occurrences in children with SCD. These infections must add to the energy toll in these children. Energy requirement, like fluid and electrolyte requirements, increases sharply during febrile episodes in children. A ten percent increase over basal needs is required for each degree ($1^{o}C$) temperature elevation above normal ($38^{o}C$). For the normal child with occasional infection and fever, this demand is readily met. In the child with SCD who has frequent infections with fever, the extra demand may be excessive. However, this demand is not limited to that required for the temperature elevation; infections also require repair in the immediate post-infection state. The energy requirements for such repairs must also be large. At present, its exact magnitude is unknown.

Similarly, vasooclusive insults resulting in micro- and macroinfarcts may involve almost any organ in the SCD patient. Such infarcts have been demonstrated in the bone, GI tract, liver, spleen, brain, and others. The energy cost of repairs of such injuries must be enormous in the child with SCD. The consequencies and the effects of all these demands on the energy sources of the body in the child with SCD have not been examined in any detail. Nor has there been any documentation of these demands and their magnitude on the energy balance in SCD patients.

Energy Requirement for TEF:

a) Thermodynamic Effects of Food--otherwise and variously termed "Specific dynamic action", "Energy of energy requirement", etc. is a requirement which is an inherent characteristic of all energy sources amounting to about 4 to 5 percent of the energy intake in normal individuals. No information exists to suggest that this requirement is altered in SCD. However, since it is dependent upon the total energy utilization of the system, it is possible that it is increased in SCD because total energy requirement in SCD appears to be increased.

Energy Requirement for Losses

Normally 4 to 5 percent of energy intake is recoverable as unusable energy in stools. This is normal energy loss. Very little energy is otherwise lost in normal subjects. However, in disease states, considerable amount of energy intake may be lost in the stool as evident in malabsorption, or in the urine such as in diabetes mellitus. The amount of energy normally lost in stools is determined by the integrity of the gastrointestinal tract and its functions. Because of the frequency with which the GI tract is involved in SCD, it is reasonable to anticipate that patients with SCD will eventually develop nutrient losses through this mechanism. As noted earlier, however, the few studies which have examined this issue have failed to substantiate any significant digestive and/or absorptive abnormalities in the gastrointestinal tracts of patients with SCD (3,6,7).

Similarly, because the renal system is frequently involved in the complications of SCD, it is reasonable to suspect that this system may eventually be a source of energy loss as well. There is no information on this matter in the literature although renal complications are not uncommon in SCD.

Energy Requirements for Physical Activity

Physical activity requires energy and the amount of energy required is proportional to the amount and degree of such activities. There is little information on the physical activities of patients with SCD and no study on the energy requirement for physical activity in SCD. Anedoctal observations of children after one year of age, suggest that children with SCD are much less physically active than normal controls. Although no proof exists, the energy requirement for physical activities in patients with SCD may be less than in normal controls.

Energy Requirement for Growth

Many growth manifestations of malnutrition appear to be present in SCD. Indirect evidence of protein-energy malnutrition, such as delayed skeletal maturation (15-17), retarded pubertal growth (18,19), retarded accretion in height and weight (15-21) and the development of eunuchoidal habitus (22,23) have been reported in patients with SCD. These abnormalities appear to be more pronounced in the male patients although females are not spared. Both sexes appear able to ultimately catch up later in adulthood. There is no consistent relationship between the growth retardation seen in SCD and any clinical or biochemical abnormalities in growth hormone production (24, 25), thyroid hormone production (14, 26) or Somatomedin C production (26, 27).

Other indirect evidence of energy malnutrition in SCD is the decreased immunologic function found in patients with SCD. Defects in T cell function associated with decreased lymphocyte counts, impaired delayed hypersensitivity, and diminished response to mitogen stimulation have been reported in patients with malnutrition (28,29). Similar findings, including decreased T4 and T8 cells, have been reported in patients with SCD (3,30,31). On the other hand, B lymphocytes are increased in both malnutrition and SCD.(29,32-34). Additional but suggestive indirect evidence of energy abnormalities in patients with SCD is the delayed onset of menstrual activities in females (19,26,35). Since weight is a significant determinant of the onset of menses and patients with SCD have significant growth delay, onset of menses appears to be delayed in these patients based on their delay in weight growth.

Few clinical parameters and laboratory studies provide direct evidence of energy nutritional state. Growth in height and weight and the relationship of the former to the latter as well as skin fold thickness have been used as indicators of nutritional status. Children with SCD, as previously noted, are frequently weight retarded, suggesting a nutritional role especially since endocrine abnormalities have not been found to be consistently related to growth retardation in SCD (14,24-27) Measurements of skin-fold thickness have not yielded consistent results (24,36-37) and the variations found appear sometimes sex related (17).

Measurements of blood sugar and serum proteins have been generally normal in SCD (4,6,27,38,39). Glucose tolerance also appears to be normal in children with SCD (38). But serum lipid values are typically reduced (31, 40, 42). As noted earlier, the most direct, although flawed, evidence for the role of inadequate energy intake in SCD is the study by Heymann, et al (3). Two of their five patients were given nasogastric feedings providing, to one patient, all of his estimated protein and calorie requirements and to the other patient 66 percent of his protein requirements and about 33 percent of his calorie requirement. These two children showed significant improvement in growth during the months they were tube-fed. In contrast, two children who were vitamin and mineral but not calorie supplemented and one child, who received only oral bedtime supplement providing 16-33 percent of his daily requirements for protein, calories, vitamins and minerals, failed to show any significant increment in weight.

Thus, in summary, it would appear that although patients with SCD seem to have adequate energy intakes and that their gastrointestinal tracts function normally, the real energy needs imposed by the disease are actually not met. There is a large energy tax burden placed on the patient with SCD by the disease. The basal metabolic rate in SCD is increased and energy losses may perhaps be similar to those of the normal controls. The energy needs for physical activities may be less than in normal subject although by how much remains unclear. Children with SCD show significant growth retardation and fail to achieve adequate heights and weights. Many who reach adulthood manage to compensate for some of the childhood deficits, although most never do catch up completely. Immune function impairment, partly due to T cell functional abnormalities with normal B cell functions, delay in onset of menarche, retarded pubertal growth, and abnormal lipid profiles seen in SCD suggest nutritional deficiency. Correction of some of these problems with vigorous nutritional intervention provides additional evidence for the role of nutrition in management of SCD. The single most important comment that can summarize our conclusion is that there is a great deal we do not know. Much remains to be learned about the energy status of the patient with sickle cell disease.

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Antioxidant Micronutients and Sickle Cell Disease

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Abstract

Increasing evidence suggests that in vivo lipid peroxidation resulting from excessive free radical production may be an important factor in determining the clinical severity of sickle cell disease (SCD). Iron in the form of membrane bound hemichrome plays a major catalytic role in the production of oxy-radicals that can initiate lipid peroxidation reactions. Dietary supplementation with micronutrients that have antioxidant properties such as vitamin E, vitamin C, zinc, selenium, and beta-carotene may decrease the severity of SCD and provide a relatively inexpensive and nontoxic form of chemotherapy. Adult SCD patients have about 50% less plasma vitamin E than normal, age-matched black controls [1,2]. Vitamin E is the major lipid soluble free radical quenching antioxidant. In addition, there is a high prevalence (86%) of retinol-binding protein complex. Deficiency of vitamin A suggests that beta-carotene (provitamin A) levels may also be low in SCD patients. Beta-carotene is a very effective singlet oxygen quencher. Selenium is an important antioxidant nutrient since it is an essential cofactor for the enzyme glutathione peroxidase (EC 1.11.1.9) which detoxifies lipid hydroperoxides (or hydrogen peroxide).

Introduction

Sickle cell disease is often cited as being the first example of a genetic disease for which the detailed molecular basis was determined. Individuals with sickle cell disease are homozygous for hemoglobin S (Hb SS genotype) which has a valine substituted for glutamic acid in the sixth position of the beta-globin chain. At present, no effective chemotherapy exists for sickle cell disease (HbSS) which is among the most common genetic blood disorders seen in this country. The medical costs, including loss of time from school and employment make this health problem one of high social and economic importance.

Despite the invariance of the genetic alteration in HbSS, the clinical severity and hematological parameters among the patients are very variable [4-7]. Some patients experience frequent vaso-occlusive crises requiring hospitalization and others remain relatively asymptomatic for long periods of time. It is obviously of some importance to understand the basis for this clinical variability since it could provide insight into possible therapeutic means of intervention. The clinical severity of the disease in a patient is most likely determined by many factors. The presence of linked and unlinked genes may modify the expression of disease and contribute to clinical variability [8,9]. Gene mapping analyses indicate that about 30 per cent of American Blacks have a deletion of one (out of four) alpha-globin genes [8]. HbSS patients with this form of alpha-thalassemia appear to have a decreased mean corpuscular hemoglobin concentration which is thought to decrease the clinical severity of the disease [8]. A high level of hemoglobin F which is influenced by genetic factors [9] is also thought to be clinically advantageous. Environmental factors such as nutrition [10,11] and exposure to pathogens also play a major role in determining the long range clinical severity of SCD.

Most recent studies of sickle cell disease have primarily been concerned with the molecular events involved in the in vitro aggregation of hemoglobin S (HbSS) molecules under deoxygenated conditions. The polymerization of deoxy-HbSS under equilibrium conditions gives rise to the sickle shaped red blood cells which are characteristic of the disease. In vitro studies of deoxy-HbS under equilibrium conditions can, however, be misleading since very recent kinetic studies suggest that the delay time for HbSS aggregation in most HbSS-red blood cells is sufficiently long so as to prevent HbSS aggregation from actually occurring in vivo [12]. Recent evidence suggests that other secondary factors may also be important in understanding the complex pathophysiology of sickle cell disease. A major factor that has yet to be fully explored is the potential relevance of in vivo lipid peroxidation and free radical damage to the erythrocyte membrane. The area of "free radical pathology" is receiving increased attention in medicine and many excellent reviews are available (13-15). In this report, we will focus primarily on the micronutrients that can potentially modulate the free radical pathology observed in HbSS patients. In particular, we will discuss the roles of some vitamins and essential trace elements such as selenium and zinc.

Lipid Peroxidation Damage In Sickle Cell Disease

Lipid peroxidative damage to the red blood cell membrane could be caused by the intrinsic instability of HbS [16,17] and by the low levels of vitamin E found in both the plasma and erythrocyte membrane of HbSS patients [1,2,18-21]. Figure 1 illustrates the role of both vitamin E and glutathione peroxidase (GSH-PX) in inhibiting lipid peroxidation. It is significant that HbSS, in contrast to normal HbAA, is a major source of superoxide radicals (•02-) which are generated by electron transfer from the heme iron group (Fe2+) in oxyhemoglobin S to oxygen followed by dissociation of the superoxide radicals [22]:

$$\underset{\text{(oxyHb)}}{\mathrm{Hb(Fe2+)02}} \rightarrow \mathrm{Hb(Fe3+)02-} \rightarrow \underset{\text{(metHb)}}{\mathrm{Hb(Fe3+)}} + \underset{\text{(superoxide radical)}}{\bullet\mathrm{02-}}$$

Safe compartmentalization of iron is critical to the health of the cell [23], and increased free radical production in SCD may be a complication of Fe overload due to frequent blood transfusions [24] or a result of accelerated intravascular hemolysis of the red blood cell [25]. Because of its relative abundance and the ease with which it changes its valency state under in vivo conditions, Fe is a major catalyst of free radical reactions [26]. Additionally, the SCD patient is at risk of major infections, especially in the first three years of life, and stimulated leucocytes produce large quantities of superoxide and hydrogen peroxide as part of the body's defense against invading organisms [26]. These free radicals are released into the surrounding medium.

The superoxide radical is detoxified by superoxide dismutases [27] which are metalloenzymes catalyzing the reaction:

$$\mathrm{2H+} + \bullet\mathrm{02-} + \bullet\mathrm{02-} \xrightarrow{\text{superoxide dismutase}} \mathrm{H202} + \mathrm{02},$$

and thereby generating hydrogen peroxide. The superoxide dismutase of red blood cells (EC 1.15.1.1) is a homodimer (MW 32,500) containing both Zn(II) and Cu(II). Although controversial, recent data support the notion that SCD patients have low Zn levels in both plasma and red blood cells [21]. Superoxide dismutase levels in SCD patients have, however, been found to be higher than that found in controls [28]. Animal studies indicate that zinc deficiency is associated with an increased production of free radicals (29). The mechanism whereby zinc deficiency promotes free radical production is not clear. There are however suggestions that Zn deficiency impairs production of the Zn-Cu containing superoxide dismutase as well as lowers levels of metallothionein. The latter inducible protein contains an abundance of thiol groups and is therefore capable of protecting against free radicals in a manner similar to glutathione [23]. It would be useful to determine if zinc supplementation in SCD patients can decrease biochemical indices of lipid peroxidation.

The presence of both hydrogen peroxide and the superoxide radical can give rise to the hydroxyl radical (•OH) via the Haber-Weiss reaction [13,14,27]:

$$\bullet 02- + H202 \xrightarrow{\text{hemichrome}} 02 + OH- + \bullet OH$$

The hydroxyl radical is a potent initiator of lipid peroxidation [13,14,27]. Transition metal ions with an open co-ordination site can catalyse the Haber-Weiss reaction. The Fe in native hemoglobin, methemoglobin or cytochromes does not have an open co-ordination site and is not effective in catalyzing the Haber-Weiss reaction. Hemichrome bound to the red blood cell membrane has, however, been found to be a very effective catalyst for the Haber-Weiss reactionn [22]. SCD patients have been found to have excessive amounts of membrane-bound hemichrome [16].

Sickle cell red blood cells spontaneously generate about twice the level of superoxide, hydrogen peroxide and hydroxyl radicals than normal red blood cells [22]. Considerable evidence suggests that SCD is associated with oxidant damage to both red blood cell membrane lipids and proteins and that this oxidative stress eventually results in premature red blood cell aging, hemolysis and anemia [30,31].

The Vitamins

The lipid-soluble antioxidants include vitamin E, beta-carotene and ubiquione. Vitamin E is considered the most significant antioxidant in plasma and in the red blood cell membrane. In a short experiment with six SCD patients, it was found that vitamin E supplementation (450 IU/day) resulted in a significant (44%) decrease in ISC count compared to pretreatment levels [18]. No long-term, double-blind, vitamin E supplementation studies have been conducted with larger numbers of SCD patients to determine if there is any significant clinical benefit.

The vitamin E content of plasma and RBCs from SCD patients is significantly lower than in normal controls [1,2,18-20]. The factors accounting for the low plasma and erythrocyte levels of vitamin E in SCD patients have yet to be fully clarified. It is possible that the decreased tocopherol content in HbSS' RBCs and plasma results from increased utilization due to oxidative stress, dietary fat malabsorption, lower dietary intake or decreased content of plasma transport molecules. Lipoproteins, particularly low density lipoproteins (LDL), are the plasma transport molecules for both cholesterol, beta-carotene and vitamin E. In the general population there is a strong positive correlation between plasma alpha-tocopherol and plasma cholesterol [32-34]. It has been reported that the low plasma vitamin E levels in SCD patients correlate with low levels of plasma lipoproteins [1,2]. These data indicate that low levels of transport lipoprotein molecules (rather than a decreased vitamin E binding capacity to lipoproteins) contribute to the low plasma vitamin E levels found in SCD patients.

In the few instances in which fat absorption has been measured in SCD pediatric patients, the results indicate a normal coefficient of fat absorption and intestinal mucosa of normal appearance [35,36]. Fat absorption data for adult SCD patients are very scarce. Vitamin E absorption tests have also been performed in SCD patients but with mixed results. In growth retarded pediatric SCD patients (with normal baseline vitamin E levels), there is a decreased vitamin E absorption in two of five children tested [35]. Chiu et al. [37] found that vitamin E absorption was reduced in pediatric SCD patients (N=10) compared with normal individuals (N=4). In the Chiu et al. study [37] the baseline plasma vitamin E levels for the SCD patients were significantly lower than in the controls. It has been suggested that the decreased level of plasma vitamin E and the decreased absorption of vitamin E in SCD patients are due to impaired bile acid secretion into the intestinal lumen [37]. This possibility seems unlikely in light of the normal fat absorption reported in these patients.

There is very little information on the plasma levels of beta-carotene in SCD patients. Beta-carotene, like vitamin E, is primarily carried in the plasma by lipoproteins. It is not known if the low levels of lipoprotein carriers found in SCD patients also correlate with low levels of beta-carotene. Beta-carotene, but not vitamin A, is an effective quencher of singlet oxygen [38]. Carotenes with nine or more conjugated double bonds are efficient quenchers of singlet oxygen but carotenes with eight or less conjugated double bonds are much less effective [38]. Singlet oxygen, because of its electronic configuration, is much more reactive than normal triplet oxygen and can initiate lipid peroxidation [39].

In vitro experiments have shown that vitamin C at a level of 0.1mM inhibits the formation of lipid peroxidation products in HbSS red blood cells [40]. However, at a level of 1mM ascorbate is ineffective and at a level of 5mM is actually a pro-oxidant [40]. Vitamin C is an effective water soluble free radical quencher but under some circumstances can act as a pro-oxidant. Recent evidence obtained from model phospholipid bilayer systems shows that vitamin C in the absence of vitamin E promotes lipid oxy-radical propagation [41]. When alpha- tocopherol is incorporated into the lipid bilayer it will reverse the pro-oxidant effect of 0.1-1 mM ascorbate and the ascorbate will also prevent alpha-tocopherol depletion [41]. Although not soluble in lipid bilayers, ascorbate appears to interact with components of the phospholipid bilayer so as to protect vitamin E [41].

The Glutathione Peroxidase System

In addition to vitamin E, the functioning of other antioxidant mechanisms may be critical in protecting the SCD erythrocyte membrane from oxidative damage. A second major protective mechanism is that of the selenoenzyme, glutathione peroxidase (glutathione-hydrogen peroxide oxidoreductase, EC 1.11.1.9). As shown in Figure 2, glutathione peroxidase is a selenoenzyme that can reduce lipid hydroperoxides (or hydrogen peroxide) to the corresponding alcohol (or water). Glutathione peroxidase utilizes glutathione as a reducing agent and produces oxidized glutathione. Oxidized glutathione (GSSG), in turn, is reduced by glutathione reductase (EC 1.6.4.2; NAD(P)H: glutathione oxidoreductase) which utilizes NADPH from the hexose monophosphate shunt as a reducing agent. The glutathione peroxidase system (Figure 2) can be influenced by a number of micronutrients such as selenium, riboflavin and vitamin B6. Plasma and red blood cell levels of glutathione peroxidase are known to reflect dietary intake of selenium [42,43]. Glutathione peroxidase activity in sickle red blood cells has been measured by a number of groups but with inconsistent results. Increased [19], decreased [28], and insignificant differences [1] have been observed between sickle cell patients and normal controls. Some of this inconsistency could be due to differences in methodology. In particular, it is important to have Drabkin's reagent present in the assay cuvette to convert any methemoglobin to the cyanomethemoglobin form. Cyanomethemoglobin, in contrast to methemoglobin, is not a substrate for NADPH-methemoglobin reductase which can utilize NADPH [44]. Sickle cell patients have increased levels of methemoglobin [18] and therefore sickle red blood cells can give a falsely high glutathione peroxidase activity as measured by NAPDH utilization.

Nutritional factors could, however, contribute to the in vivo variability in the activity of glutathione peroxidase. From animal studies, it is also known that vitamin B6 is involved in the metabolism of selenomethionine to supply selenium for glutathione peroxidase [45]. In the presence of vitamin B6 (pyridoxine HCl), selenium in the form of selenite or selenomethionine is equally effective in promoting the synthesis of glutathione peroxidase. In the absence of vitamin B6, selenite but not selenomethionine is effective in promoting glutathione peroxidase synthesis. About 10 % of SCD patients appear to be deficient in pyridoxine as measured by erythrocyte aspartate aminotransferase activity coefficient [46]. In addition, SCD patients show a marked riboflavin deficiency as measured by a decreased erythrocyte glutathione reductase activity [46,47] and by a high erythrocyte glutathione peroxidase activation coefficient [46]. Glutathione reductase requires flavin adenine dinucleotide (FAD) as a cofactor. Riboflavin deficiency in SCD patients also appears to decrease the activity of glutathione reductase (a riboflavin dependent enzyme). Pyridoxine phosphate oxidase is responsible for the synthesis of the active form of vitamin B6 (pyridoxal phosphate) by the oxidation of pyridoxamine and pyridoxamine phosphate. Pyridoxine phosphate oxidase deficiency due to poor riboflavin status appears to limit the availability of pyridoxal phosphate in SCD patients [46].

A decreased activity of glutathione reductase (due to riboflavin deficiency) and a decreased ability to synthesize glutathione peroxidase from selenomethionine (indirectly due to poor riboflavin status) could certainly contribute to an impaired glutathione peroxidase system in SCD patients.

It may well be that SCD patients require a significantly increased intake of selenium compared with normal controls. In a small group of SCD patients (N= 12), it was found that plasma glutathione peroxidase activity was significantly lower during epidoses of vaso-occulsive crises and that the overall clinical severity of SCD inversely correlated ($p < 0.02$) with red blood cell glutathione activity [48]. In this study, the activity of red blood cell or plasma glutathione peroxidase in the SCD patients was not significantly different from the values observed in age-matched black controls [48].

General Discussion

It is significant that the degree to which sickled red blood cells adhere to vascular endothelium strongly correlates with the clinical severity of sickle cell disease [5-7,49]. RBC adherence to capillary endothelium may be the primary cause of vaso-occlusive crises in sickle cell patients. Sickle cell adhesion is likely to involve interaction between the RBC plasma membrane and the capillary endothelium. Much attention has recently been focused on the abnormalities found in the sickle cell plasma membrane [7,40]. Many of these membrane abnormalities may be caused by in vivo lipid peroxidation. Oxidative damage to the HbSS RBC membrane may play a major role in the formation of irreversible sickle cells (ICSs) [40].

Irreversible sickle cells maintain an abnormal sickle cell shape under both oxygenated and deoxygenated conditions, and high levels of ICSs may play a major role in the autosplenectomy that occurs in young children with SCD. Autosplenectomy in SCD children is thought to result in damage to the immune system and an increased susceptibility to infection. It has recently been demonstrated that HbSS RBCs that contain a high proportion of ISC also contain a high level of endogenous products of oxidative damage [22]. The proportion of ISCs can vary from 10% to 50% from one SCD patient to another. The biochemical basis for this wide variability in ISC count is not known but may involve differences in the dietary intake of antioxidant micronutrients.

The crucial importance of vitamin E is exemplified by the recent findings of accelerated red blood cell senescence in vitamin E deficient animals [50]. RBC senescence is accompanied by the appearance of a "neo-antigen" on the cell surface which initiates IgG binding and removal of the RBC by phagocytic cells [50]. The "neo-antigen" has been shown to be a proteolytic product of "band 3" which is an integral membrane bound protein that functions as the anion channel for chloride and bicarbonate anions [51].

Recent work also suggests that RBC removal in sickle cell disease involves increased IgG binding [52-53]. Red blood cells from normal individuals have less than 25 IgG molecules per red cell whereas 39 of 62 SCD patients had a mean of 195 and a maximum of 890 molecules of IgG per red cell [53]. In vitro studies by Hebbel and Miller [31] have shown that sickle red blood cells are ingested five-fold more than normal red blood cells by macrophages. Furthermore, the interaction between macrophages and sickle red blood cells is partly dependent upon IgG binding since it can be partially blocked by Fc receptor blockade. Treatment of normal human red blood cells with MDA or with peroxide results in an enhancement of their phagocytosis by macrophages [52]. Sickle red blood cells spontaneously generate about twice the level of MDA than normal red blood cells [52]. The results of both human and animal studies are significant because they suggest that oxidative damage to the sickle red blood cell membrane is linked with an immunological mechanism for red blood cell removal. Hebbel and Miller have estimated that two thirds of red blood cell hemolysis in SCD can be attributed to the interaction between sickle red blood cell and the reticuloendothelial system [52]. The molecular mechanism whereby oxidative damage to the red blood cell membrane results in the appearance of senescent cell antigen is not clear. Nor is it known whether or not other deficiencies in micronutrient modulated antioxidant mechanism (e.g., selenium dependent glutathione peroxidase) would also result in increased appearance of senescent cell antigen.

Deficiences of micronutrients that modulate antioxidant defense mechanisms may play an important role in determining the clinical severity of sickle cell disease. The information presented in this report suggests that both the vaso-occulsive and the hemolytic component of SCD can be influenced by antioxidant nutrients.

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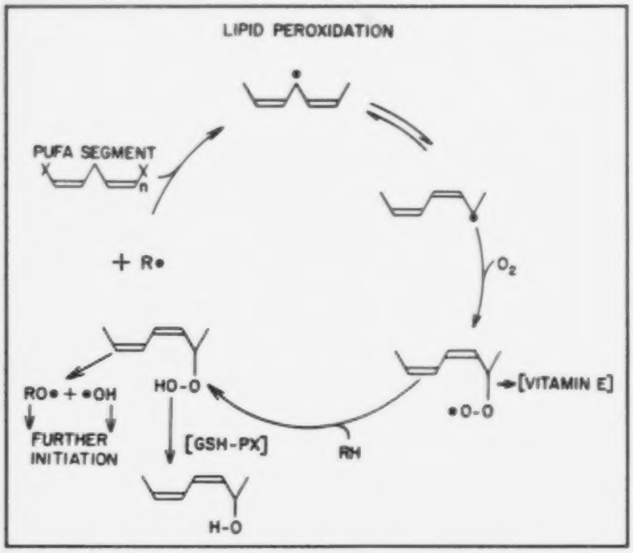
LIPID PEROXIDATION
PUFA SEGMENT
X
X
n
+ R•
O_2
[VITAMIN E]
•O-O
RH
HO-O
[GSH-PX]
RO• + •OH
FURTHER INITIATION
H-O

Figure 1— The possible role of vitamin E and glutathione peroxidase (GSH-PX) in the inhibition of lipid peroxidation. Vitamin E most likely quenches the lipid peroxy radical since the half life of this radical is comparatively long. Glutathione peroxidase reduces fatty acid hydroperoxides which might otherwise be catalytically decomposed by heme compounds into additional free radical propagation reactions. The free radical initiator of lipid peroxidation is probably derived from oxy-radicals (see text).

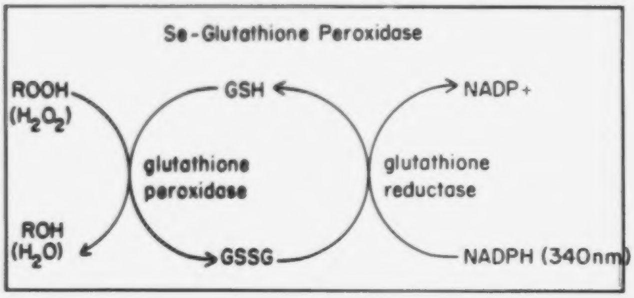
Se-Glutathione Peroxidase
ROOH
(H_2O_2)
GSH
NADP+
glutathione peroxidase
glutathione reductase
ROH
(H_2O)
GSSG
NADPH (340nm)

Figure 2—Selenium dependent glutathione peroxidase can utilize either lipid hydroperoxides (ROOH) or hydrogen peroxide as a substrate. Oxidized glutathione (GSSG) is reduced by glutathione reductase.

Sickle Cell Anemia: Discussion

Reid, C.

My question is addressed to Dr. Turner and it relates to Dr. Stone's presentation on lipid peroxidation. You mentioned earlier that tissue hypoxia and necrosis are present in patients with sickle cell disease, and you showed slides of leg ulcers in these patients. Can you comment on the potential benefit of using hyperbaric oxygen to decrease hypoxia and enhance wound healing in these patients?

Turner, E.

This has been tried in patients, and it has not been successful clinically.

Natta, C.

It seems from studies we have done that vitamin E, selenium and carotenoid levels are low in sickle cell anemia. These adversely affect the whole antioxidant system.

Stone, W.

Dr. Natta, I recollect, examined a number of patients given 450 international units of vitamin E and found that the number of irreversibly sickled cells did decrease statistically. I must however point out that we are dealing with different species of free radicals, and it may be that manipulating only one antioxidant nutrient may not tell us the whole story. Someone has raised the question of the effect of vitamin E deficiency on the arachidonic acid cascade. I am not aware of any studies that have examined this in sickle cell anemia.

Carter, J.

In terms of modalities of treatment, several points come to my mind. A number of us have been promoting clinical trials with a supplementary mixture of antioxidants, e.g. vitamin E, ascorbate and selenium, as well as the use of calcium channel blockers. The other avenue is the use of chelating agents. Dr. Stone mentioned iron as a principal catalyst of lipid peroxidation. The same can be said of copper.

Natta, C.

I am not at all sure of any connection between increase in polyamines and free radical pathology. Our studies have however shown that some patients with sickle cell anemia show a 3-5 fold increase in the polyamines, putrescine, spermidine and spearmine in the red cells. The latter decrease in amount if the patients are given vitamin B6 (50mg twice daily over a period of 6 months). We are now examining the combined effects of vitamins B6 and E.

Enwonwu, C.

From my review of the pertinent literature, it will appear that we are probably attaching exaggerated importance to the increased production of polyamines in sickle cell disease. To my mind, increased polyamine production is a feature of accelerated growth, and tissue turnover, and you see the same thing in the developing brain.

Natta, C.

From our findings in asthmatic patients supplemented with vitamins B6, I am not at all sure that increase in polyamine levels is a feature of only rapidly dividing cells.

Carter, J.

The second question I have with regard to free radical pathology relates to the concept of reperfusion injury which is now well defined, at least, in terms of myocardial ischemia. Does such a situation apply to sickle cell disease crisis?

Stone, W.

The only literature that addresses this area that I have seen is from Dr. Demopoulos in New York, and this is a key component of his hypothesis with respect to the pathogenetic features of sickle cell disease.

I want to address the other important point which you raised earlier. One has to be very cautious in using iron chelators. Iron can still be a very strong pro-oxidant when some of its coordinations are blocked. For example, in my laboratory, if we want to oxidize things, we in fact use EDTA-iron which turns out to be a better oxidation catalyst than iron alone.

Carter, J.

I wish to make a couple of comments about the increased protein turnover in sickle cell anemia. We should start when the victim is very young to give extra calories and nutrients. Studies from the USDA laboratories in San Francisco indicate that one of the best ways to increase nutrient density of foods consumed by children is to reduce the fat content.

Kinnard, M.

This question is directed to Dr. Enwonwu. I must first apologize for the tortuous nature of my question. I want to make the observation first that I am troubled by the disparity in the number of AIDS cases reported in blacks as opposed to non-blacks. Now let me get back to your presentation this morning about the amino acid histidine not being well absorbed in sickle cell anemia. Candidiasis is a common opportunistic infection in AIDS, and histidine-rich proteins are reported to exert protective effect against Candida albicans. Could the deficit of histidine in sickle cell anemia be a major handicap?

Enwonwu, C.

I don't believe I said that there is impaired absorption of histidine in sickle cell anemia. What I did mention was the strong possibility of increased dietary requirement for this amino acid in this genetic disorder based on the following argument. Hemoglobin is rich in histidine, and accelerated breakdown of this protein in sickle cell disease may promote increased loss of histidine due to inefficient re-utilization plus the fact that the renal conservation of this amino acid is not as efficient as that of other amino acids. Certainly, inadequate histidine status in sickle cell anemia will pose some problems since this amino acid, which is now considered indispensable for the human, features prominently in erythropoiesis. On the question of the role of salivary histidine-rich proteins as effective fungicidal agents particularly against C. albicans, I believe the relationship is still speculative although there are strong suggestions that imidazole compounds have anti-fungal effects.

South, M.

In terms of severity, the infections in sickle cell anemia are nowhere comparable to the situation in AIDS. Sickle cell patients do not have chronic progressive candidiasis as seen in AIDS. They don't die from ordinary viral infections, nor do they have pneumocistis carini. It seems to me that the major problem in respect of infections in sickle cell anemia results from defective phagocytosis. The phagocyte uses a lot of energy for intracellular killing of microorganisms, and the sickle cell patient has a very high energy need.

Reid, C.

I think you have made a good point. Clearly then, nutrition has an important role in the management of sickle cell anemia.

Margen, S.

In terms of increased protein requirement in sickle cell anemia, I think it is more profitable to look into the needs for specific amino acids. Also, when I hear that children with sickle cell anemia are not growing well, that information immediately tells me that they are receiving inadequate food.

Turner, E.
Data on retrospective studies coming out from the University of Southern California suggest a significant decrease in both hypertension and myocardial disease in blacks with sickle cell disease compared with those without.

Stone, W.
I don't believe that because serum lipid levels are lower in sickle cell patients than in normal individuals, one can safely assume that the former are less susceptible to cardiovascular disease. The LDL/HDL ratio is probably a better predictor than just the cholesterol level. There is also some evidence that LDL and HDL levels are independent predictors of cardiovascular disease risk and that a low HDL level is beneficial.

Anonymous.
I have heard of use of dietary cyanates in treatment of sickle cell anemia. Will any of the panelist care to comment on this.
Enwonwu, C.

There are many substances already shown to retard RBC sickling in vitro. These include urea, cyanate, and others. The levels required are not applicable in vivo.

Reid, C.
Studies have shown that in vivo use of large mounts of cyanate produces neurotoxicity, cataracts and other health problems. We do not recommend it for clinical management of sickle cell patients.

There are enough challenges in this area to keep us scientifically occupied well into the next century. A recent study we have done shows that small doses of oral penicillin prophylactically could prevent pneumoccocal infection in sickle cell patients. So, for the first time we do have a good reason for screening newborns and for instituting prophylactic regimen in the first 4 months of life.

Finally I wish to say that this workshop has heightened our awareness about sickle cell disease in general, and specifically about the potential role of nutrition in its management. While it has not given us a clear-cut consensus or direction for research, it has certainly provided some very challenging perspectives regarding needed basic and applied research.

SIXTH SCIENTIFIC SESSION

Local and International Health Issues

CHAIRPERSON:

Wallace T. Williams, Ph.D., R.D.
Dean, College of Fine and Applied Arts
University of Akron
Akron, Ohio

Nutrition Issues In Tennessee

Lee Fleshood,
Nutrition and Supplemental Food Programs
Tennessee Department of Health and Environment
Nashville, Tennessee

Introduction

First, my congratulations and thanks to Meharry, Dr. Enwonwu and the Nutrition Center staff for a very well planned workshop and the opportunity to participate. The working relationships between staffs of the Department and Meharry extend over many years and are highly valued. I personally have appreciated the opportunities to work with the late Dr. Ed High, Dr. Les Falk, Dr. Hank Foster and other Meharry staff over the years. We are excited about the positive impact the Nutrition Center is having and will have on state, national and international nutrition needs. My task is to share with you some "Nutrition Issues in Tennessee" that are being addressed by public health staff. Over the past two and a half days we have heard an excellent review of the current scientific and behavioral research which provides direction and guidance for health providers. Now we come to "where the rubber hits the road," so to speak, or "where we try to do the most, for the most, for the least" through public health services.

My colleage, Dr. Smith, will be discussing some of our concerns "Beyond WIC". The Nutrition and MCH units share many mutual agenda in the delivery of maternal and child health services that impact on health and disease. There are many more topics that we would like to discuss with you than either of us have time to cover, therefore we have attempted not to be repetitive in our remarks. Over the past 14 years I've seen Tennessee public health nutrition services come a long way. On the other hand I realize we've got a long way to go. For this first conference I would like to discuss with you:

Outline

1. Mission and our county nutritionist concept
2. A little philosophy concerning nutrition and tools for health service delivery
3. The importance of and some results from Pediatric and Pregnancy Nutrition Surveillance Systems, particularly data relating to Iron Deficiency Anemia, Infant Mortality, and Low Weight Births
4. Tennessee Infant Formula Rebate Project

This means some very important nutrition/nutrition related issues and projects such as behavior risk factors, surveillance, breastfeeding, intensive nutrition education for prenatals, hunger/homeless, nutrition for the elderly, and weight management fitness information will have to wait for another time.

There are 95 counties in the state and over 600 miles from the east to the west borders. The geographic and demographic differences provide many challenges to statewide service providers. For example, while the states' 4.8 million population is 84.0% white and 16 percent other, Memphis, along the Mississippi Delta, is about 50% black, contrasted with some counties in mountainous Appalachian East Tennessee where the black population is less than 1%. Five counties have populations greater than 100,000 while the other 90 are mostly rural. These differences must be an integral part of needs assessment and planning when implementing services.

Mission and Organizational Structure

The mission or purpose of Nutrition and Supplemental Food Programs, simply stated, is to reduce nutrition related morbidity and premature mortality. This means our priorities include attention to the needs of Tennesseans from our very young to our senior citizens.

Services are provided through 13 Regional Offices and the 95 Local Health Departments. Regional Nutrition Directors are responsible for developing nutrition plans, and for delivery of direct services. Well trained nutrition staff are necessary to ensure quality services and to support our other professional staff.

Across the state we have 126 nutrition positions funded with a combination of WIC, Commodity Supplemental Food (CSF) program, state local or other funds. Currently 88(79%) of the positions are funded with supplemental food monies.

County Nutritionist Concept

We promote and encourage what we call the "County Nutritionist Concept", which simply means that the Public Health Nutritionist is based in the county, functions as part of that county health care team, and has total responsibility for planning and implementing nutrition services in his or her county. We have defined three levels of nutrition services, depending on the extent to which a nutritionist is available and functioning within a given county (Table 1). We believe Level 3 to be the most beneficial one for the patient, county and the nutritionist. It certainly reduces turnover in dynamic staff who can easily burn out from working only with WIC patients.

Support of this concept requires us to expand our funding base. We are pursuing four primary routes other than supplemental food programs to meet the needs of the community: 1) Staffing - encouraging other health programs that benefit from nutrition services such as primary care and programs that serve the handicapped and chronically ill to fund part or fulltime nutrition staff. 2) Grants - in the past year we have received a DHHS Breastfeeding SPRANS Grant, a Diabetes Grant, a Nutrition Education Grant through USDA, and a Pregnancy Nutrition Surveillance System cooperative agreement through CDC in Atlanta. Each grant added to the funding base by supporting nutrition FTE's (fulltime equivalences). 3) Local funding - recently one county in the Southeast Region of the State agreed to fund "their own nutritionist: to work 100% of her time in their county. This agreement came only after the WIC funded nutritionist served in varying degrees as a county nutritionist for four years. 4) And finally, fee-for-service activities.

Philosophy: Nutrition As a Component of Health Services.

I would like to share with you a little philosophy with five major points:

1. We (nutritionists in Tennessee) believe that a strong nutrition component/unit is essential to effective federal, state and local public health programs.
2. Most administrators know that a strong nutrition component is obtained by recruitment and support of well trained, creative, motivated and dedicated nutrition staff.
3. Support means staffing nutrition components within the programs, adequate salary and career ladder, encouraging continuing education/training, promoting comprehensive nutrition services for the life cycle, and providing critical tools.
4. WIC, in a sense, is a tool (or outstanding resource) that can be used as a base or the vehicle to build a quality comprehensive nutrition program. When viewed from a broader perspective, WIC, along with immunizations and prenatal care, is/can be the foundation of maternal and child health services to low income persons. All states and nearly every county (and

P.H. clinic) in the nation have WIC available, as that vehicle. The Tennessee WIC Program currently receives $37 million to serve approximately 77,000 women, infants and children. Twenty percent ($7.4 million) provides health screening, nutrition education and the food delivery system. The remaining 80% provides a monthly supplemental food package. Tennessee has received national recognition for innovative programs and projects connected with WIC.

5. High priced tools like WIC, Early Periodic Screening, Diagnosis and Treatment (EDSDT) and others require adequate steering at all levels to be efficient and effective. I can not over emphasize the importance of relatively low cost tools like pediatric and pregnancy surveillance systems as a very high priority, after getting that qualified local nutritionist in place.

To some extent all of us attempt to be accountable to two questions:

1. Are we doing things right?
2. More importantly, are we doing the right thing?

Extensive regulations, standards, and audits can get us so bogged down in working on the first question that we lose sight of the second. I believe that tools like pediatric and pregnancy surveillance are essential to maintaining significant balance between the two questions. They also provide a way to monitor our progress towards obtaining desired health outcomes, like those stated in the 1990 objectives for the nation. Optional effectiveness of these systems requires that they be owned and used by staff at all levels, especially at the clinic level.

Pediatric Nutrition Surveillance

In addition to assisting with quality assurance problems, state and local pediatric nutrition surveillance provides extremely valuable longitudinal data that have not been possible to obtain from multimillion dollar national surveys. In 1973, Tennessee was one of the first five states to participate in the pediatric nutrition surveillance system promoted by the Centers for Disease Control (CDC). The system collects height, weight and hematocrit measurements for consolidation, analysis, and feedback to the clinics. These measurements are the basis of physical assessment for children and tracking their growth and development. Therefore, attention must be given to equipment, training and follow-up activities (1-5).

CDC assists multiple states in collecting pediatric growth and hematological data from low-income children enrolled in various public health programs, including WIC, and EPSDT, Head Start and maternal and child health clinics (6). The next 2 slides include nutrition surveillance data collected from Arizona, Kentucky, Louisiana, Montana, Oregon, and Tennessee from 1975 to 1985.

Figure 1 represents over a million hematocrits (hcts) collected from over 400,000 children in six states. Details of the study are available (7). Anemia is defined as a hct. below 32%. For the six states, the overall prevalence of anemia has declined from 7.8% in 1975 to 2.9% in 1985. Figure 2, demonstrates that in the earlier years blacks had a higher prevalence of anemia, but the difference between blacks and whites narrowed as the rate of anemia declined. Racial distribution of the data breaks down to 71.8% white and hispanic, 20% black, 7.5% American Indian.

We are pleased that CDC selected Tennessee to complete a major data linkage project. The following data are linked sets from two sources. One data set consists of records of Tennessee pediatric WIC program participants from 1975 to 1984 and includes date of birth, sex, race, height, weight, hematocrit measurements, and birthweight reported by parents. The other are birth certificates provided by our Center for Health Statistics. These records contained information on prenatal sociodemographic variables, like prenatal care and birthweight, in addition to the child's name and date of birth.

In this study (8) we looked at height. for age, weight. for age, weight. for height., and hematocrit measurements. Initial or pre-enrollment screening to WIC programs were used as the "Non-WIC" controls. Most of these children were measured at the time of enrollment in the WIC program. However, the measurement on the day they enrolled actually reflects the pre-enrollment health status. In essence, the WIC and Non-WIC comparison is based on comparing children not on WIC previously (at initial visit) with those on WIC six months or longer.

Socioeconomic Stratification (SES)

Trends were determined for children from different SES groups enrolled in WIC. Family SES for these children was based on three socio-demographic risk factors from the birth records: (1) mother's age 17 or less at the child's birth; (2) mother's education less than 12th grade; and (3) mother unmarried. We defined low SES as those whose families had two or three risk factors, intermediate SES as those families with only one of the risk factors, and higher SES as those families which had none of the risk factors. We further divided the SES group into two subgroups based on whether or not at least one of the parents had more than a high school education. This procedure of defining SES background resulted in four SES groups ranging from high SES (group 1) to low SES (group 4). You can see from Figure 3 that en though the SES composition remained stable from 1975 to 1984, the prevalence of anemia has declined significantly within each SES group.

Figure 4 shows the distribution of all Tennessee children as well as the distribution of WIC-enrolled children among the four SES-risk groups. It is evident that by contrast, only a small fraction of the children in the lowest risk group (group 1) were enrolled in WIC. The overall distribution of SES-risk of WIC-enrolled children is skewed toward the higher groups when compared with the distribution for all Tennessee children. These findings indicate that the Tennessee WIC program is preferentially serving children with higher risk backgrounds. There were no significant differences (8) in Weight. for Age or Weight. for Height. Comparison of rate of anemia for children 12-17 months between WIC and non-WIC is dramatically shown in Figure 5. We also found WIC had a dose response effect on anemia which I will not cover here (9).

Summary Statement about The Pediatric Nutrition Surveillance System

These findings indicated a positive association of WIC with better height (Figure 5A) and hematologic status after controlling for age and SES and further suggested that the greatest benefits of the program occurred among those of the lowest SES status within the WIC-eligible population. No association was observed between WIC and childhood weight status (Figures 5B and 5C).

One fundamental difference of this study from previous evaluations is the ability to control the background of both WIC and control children - stratification by SES groups with verification of birthweight and prenatal care characteristics. The birth certificate records help to ensure the comparability between WIC children and their control. It is possible there are other factors which may account for the differences observed and not available on either the WIC or birth records, but the family characterization for this study is as complete as any population based data-set this size can provide. One reason for the observation of a better trend in the WIC children may lie in the power of the study - significantly larger sample size than many previous evaluations. Another reason which may contribute to these observations is the separation of the population by different SES groups. We demonstrated in this study that there was wide SES distribution among WIC participants and nutritional outcome differences between WIC and control children appeared to be greater at the lowest SES end. This variation of potential WIC impact across SES groups would reduce the chance of detecting differences between WIC and control children if children of all SES backgrounds were grouped together. In fact, we suspect that the national WIC evaluation conducted by Rush et al, failed to observe strong trends in health outcomes because of the variation of SES background among study as well as control subjects and a possible different SES mix between their study and control groups (18).

It is critical to have HANES and for WIC to be evaluated on a national basis. We also very much need the national Nutrition Monitoring legislation that has been pending for three years. I also believe it is extremely important that each state and every clinic receiving WIC and MCH funding have nutrition and pregnancy surveillance systems. Not only are these systems capable of providing data that national evaluations have yet been unable to provide, but they are also absolutely essential for quality control to improve care delivery. The value of the pediatric surveillance system for quality control has time and again been demonstrated over the past 14 years in Tennessee (10-14).

Infant Mortality (IM) and Low Weight Births (LWB)

Figure 6 illustrates the dramatic reduction in IM in Tennessee over the past 20 years and a disturbing stability (15) in the percentage of LWB for the same period.

Provisional data for 1986 show the infant mortality rate per 1,000 live births at its lowest level ever:

	Overall	Blacks	Whites
1985	11.3	19.4	8.8
1986	11.0	18.3	8.7

However, despite a continual drop in both White and Black infant mortality over two decades, Black infant mortality has continued to be twice that of Whites. Further, there has not been a major decrease in Low Weight Births (LWB) in that period, indicating a need for additional preventive and outreach services (see Figures 7 & 8).

Although LWB is rarely given as the cause of death on birth certificates, the majority of Tennessee infants who die each year are LWB. In 1985, 63.2% of infants who died in Tennessee were LWB. We should note that Tennessee's infant mortality and LWB rates may be artifically high since infants that weigh less than 500 grams were often counted as live births.

WIC has been shown to have a positive impact on birth weight (16-18) as described in previous presentations. Briefly I will discuss two ocess indicators that show how well WIC services are being used by pregnant women, especially those who actually have a LWB and why I think we are not having any impact on statewide LWB statistics, as yet. Before I do that, let's look at teens in the state:

- Teenage pregnancy is going down but remains much too high (Figure 9).
- 15 to 17 year old blacks are twice more likely to get pregnant than whites (Figure 10).
- 10 to 14 year old blacks are almost 10 times more likely to get pregnant (Figure 11).
- And there is a great difference in the rate of illegitimate births which continues to increase for both white and other races (Figure 12).

School health projects and several other efforts are underway to address these problems.

Indicators of Outreach to High Risk Prenatals

There are other indicators but the two I want to discuss with you address the question of outreach to the very high risk women. The indicators can be defined by questions:

1. How many women (or what percentage) actually having a LWB received WIC as part of her prenatal care?
2. How early are the women getting into care?

Our hypothesis is that greater improvements in these indicators must occur in communities across Tennessee before significant reduction in statewide LWB will be observed. Figure 14 provides data from 92 counties which operate only the WIC program (CSF or CSF and WIC are operated in three counties and these data are not available from the CSF data system). Over the last four years we have had a steady increase in the percentage of prenatals served. In 1986, 14,145 women were seen as prenatals and another 8,853 infants were seen, whose mothers did not get health department prenatal care or WIC. Looking at the cup half full, 68% of the women who actually had a LWB received WIC services during the prenatal or postnatal period. Looking at it half empty, two-third of the women we would most like to see receive the benefits of WIC did not. Out of the 3,188 women in Figure 14 who had a LWB delivery in 1986, 34.1% (1,087) did receive WIC some time during their pregnancy. However, 63% of the 1,087 received WIC for less than 20 weeks before delivery. This means that among the 3,188 high risk LWB women only about 10% began receiving WIC in the first trimester. We can not expect prenatal services and WIC to significantly impact statewide LWB statistics when only 10% of the women most in need of services are receiving them early in their pregnancy. Data from four regions (representing 36 counties) show that 75% or more of the women with LWB received WIC as prenatal or postnatal patients. Therefore, we believe that a substantial number of the one-third that did not receive WIC as prenatal or postpartum clients are eligible. As an example, eleven counties in the Southwest Region of the State saw 87% of their LWB either as prenatal (54%) or postpartum (33%) in 1986. This also shows how LWB is associated with low SES since such a large percentage of these patients are eligible for WIC.

Summary of Statement about Pregnancy Data

Significant reduction in Tennessee's LWB over the next five to ten years will require a tremendous amount of cooperation from public and private concerns interested in improved pregnancy outcomes. Prenatal services and the WIC Program have an important role to play. We must do a better job in preventing LWB amount the women we do get into care as well as get more of these high risk women into care early. Some important needs for the latter include:

1. Aggressive referral systems and positive experiences of friends and relatives who have used WIC services.
2. An environment where all parties (care providers, and recipients of services) periodically review acceptability and accessibility. Areas of review might include location, time of clinics, appointment systems, length of waiting, and surveys of users' and non-users' opinions.
3. Local and state review of key outcome, outreach, and service indicators.

Tennessee Infant Formula Rebate Project

I will close by giving you a brief overview of the infant formula rebate project that will provide additional funding to pursue outreach efforts to serve more high risk women and infants.

Tennessee is the first state to field a team capable of implementing a WIC infant formula rebate project through a voucher delivery system. The project will save $4 million WIC dollars in fiscal year 1988. To do justice to the experience that took over three years in planning and start up requires much more time than I have left. I do feel a brief history of the rebate project will be of interest to you.

History of the Rebate Project

In a sense, the rebate project had its beginnings in Tennessee in 1980 when we hired our first fulltime merchant coordinator. One of this person's major job responsibilities was to monitor differences in the food package and reduce costs while maintaining a quality food package.

In the early 1980's, we felt the cost of infant formula was increasing too fast. WIC pays for one-third of the infant formula purchased in Tennessee. Although the cost to the state was indirect through a voucher system, we began to explore alternatives for obtaining a lower price without disrupting our voucher delivery system.

During this time infant formula prices continued to climb and prices more than doubled in eight years (as seen in Figure 14), while prices of other WIC foods increased only 20-30%. It was apparent that our expressed concerns about price increases made no impact on pricing policy by the formula companies.

We learned that any substantial price reduction would have to come from the manufacturer who accounts for 90% of our $1.53 cost (August 1987) per can. The wholesale/retail distribution system accounts for only about 10% of the costs (Figure 15).

Officially the rebate project was initiated in the summer of 1984 with a change in the state WIC rules. The change allowed the WIC program to contract with one manufacturer as the primary provider of infant formula in exchange for a rebate.

Approximately 18 months later, December 16, 1985, an invitation to bid was released. It took that long to get everybody on board because no one had ever done anything like what we were proposing in Tennessee or in any other state. By everyone I mean the various units within our own Department and the Departments of General Services, Finance and Administration as well as the Offices of Treasurer, Comptroller, Attorney General and Governor. To be successful the project required a lot of support and a team effort at all levels.

We received supporting media coverage in the local newspapers. Bids were due on January 22, 1986. A January 6, 1986 pre-bid conference was attended by representatives of the three major formula companies, Infant Formula Council and their legal counsel. You've seen cartoons with, "You know you're in trouble when...!. Well, you know you're in trouble when potential bidders show up to your pre-bid conference with their lawyers. Sure enough, within the week, USDA and Tennessee were hit with 22 pages of legal rhetoric. At last minute USDA did not want us to open the bids without holding additional hearings. Since the state was not required to act on any bids, we opened the two envelopes received only to find two companies formally declining to bid. Again, media coverage was supportive. However, it was back to the drawing board. We had additional meetings with formula company representatives and held public hearings. Lacking substantive reasons to do otherwise, another invitation to bid was sent out July 1986. Wyeth Laboratories was the only company to bid. The initial bid was $.40 per can. Again we received positive media coverage. After an intense period of "getting ready" we began to phase in Wyeth only vouchers in March 1987 and became fully operational June 1, the starting date of the 20 month contract. The contract with Wyeth stipulated that the rebate would go up simultaneously and in the same amount as any price increases. So currently we receive $.56 per can of SMA and $.58 per can of Nursoy.

Under this system mothers with WIC vouchers are permitted to buy only the milk or soy based formula designated on the WIC vouchers. Exceptions are made on the basis of medical need (infants who do not tolerate the designated brand are able to receive other formulas with a prescription from their physician). Currently prescriptions for standard formula are running approximately 14%, which we expect to decline to less than 10%. We plan to use the rebate to increase participation by 8,000 eligible people, if we receive additional administrative funds.

Thus far, the two major disappointments have been the lack of administrative funding support from USDA and continuous misinformation campaigns by some formula representatives.

To date USDA has not provided Tennessee any additional administrative funds in support of the project. Once again the media have been supportive. There is legislation pending in congress that would allow states to use 10-20% of the money saved from competitive bids for the administrative cost of increased participation.

The second disappointment has been a decline in the credibility of some infant formula representatives who have been untruthful and unethical in describing the Tennessee rebate program to others. When we have been able to tell our side of the story the vast majority of those interested have been supportive.

It has been extremely gratifying to know that Tennessee's efforts have facilitated alternatives available to states for lowering infant formula costs and serving hundreds of thousands additional eligible people with WIC benefits, while maintaining their voucher delivery system.

Acknowledgment

Presentations like this would not be possible without the assistance and support of many co-workers in the Department of Health and Environment, especially those seeing patients each day in regional and local clinics. The Pediatric Nutrition Surveillance data and discussion are also by R. Yip, Nancy J. Binkin and FL. Trowbridge.

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Table 1

DEFINITION OF
LEVELS OF COUNTY/CLINIC NUTRITION SERVICES
TENNESSEE DEPARTMENT OF HEALTH AND ENVIRONMENT 1984

Level 1*

WIC, plus nutritionist has responsibility for all the nutritional requests from the county and provides services to more than one program which should include one of the following: EPDS&T, Family Planning, Prenatal or Preventive Health Counseling. County health department personnel clearly understand which nutritionist to contact with their requests.

Level 2*

WIC and Level 1, plus a nutritionist providing services for all health department programs. In addition to Preventive Health Counseling, other revenue generating** nutrition services are available to the county via the Regional Office (e.g., Weight Management/Fitness Program; Cooking-for-Health; contracts with Departments of Education, Corrections, Human Services, Mental Health and Mental Retardation, or other state and private agencies).

Level 3

Level 2, plus nutritionist has responsibility for developing and implementing the total trition program plan for the county (with the assistance and guidance from the Regional Nutrition Director or his/her designee). Nutritionist's work station is a county.

*Plus marks may be used to indicate services beyond the stated level and approaching next level.

**Revenue Generating Activity is defined as revenues generated from third party payments or from resources other than those provided from Nutrition/WIC Services or other related programs in Nashville

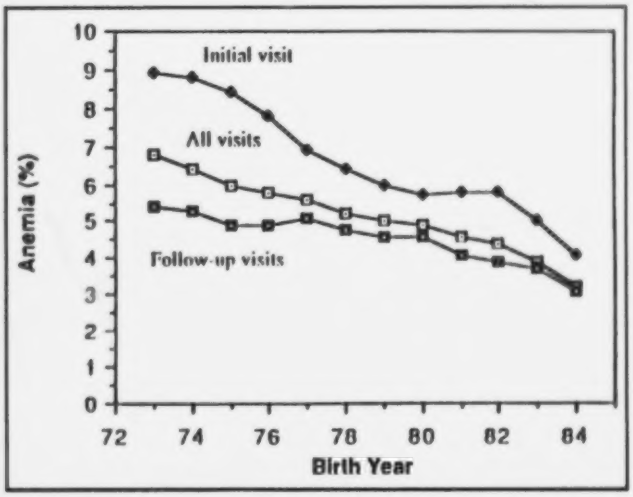


Figure 1: Prevalence of anemia for each birth year cohort, six selected states in CDC Pediatric Nutrition Surveillance System. The top line represents prevalence for children seen at initial visit only, the bottom line represents the prevalence for anemia of children seen at follow-up visits, and the middle line represents all visits combined.

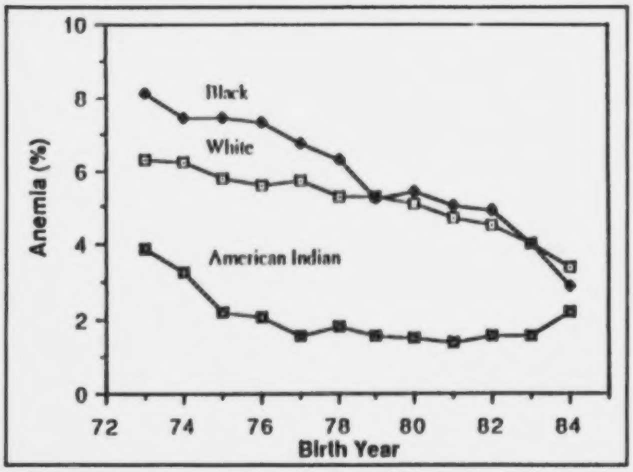


Figure 2: Race-specific prevalence of anemia based on birth year cohort, six selected states in the CDC Pediatric Nutrition Surveillance System.

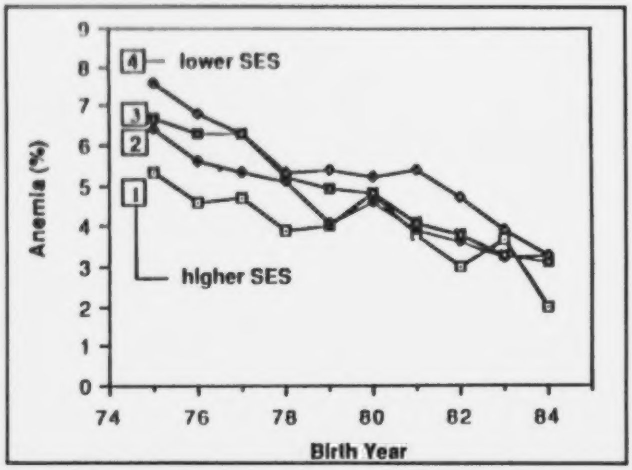


Figure 3: SES-specific anemia trends of Tennessee children enrolled in WIC program, 1975-1984. All SES groups showed a decline in prevalence of anemia even though the lower SES groups have higher anemia prevalence in a given year than the higher SES groups.

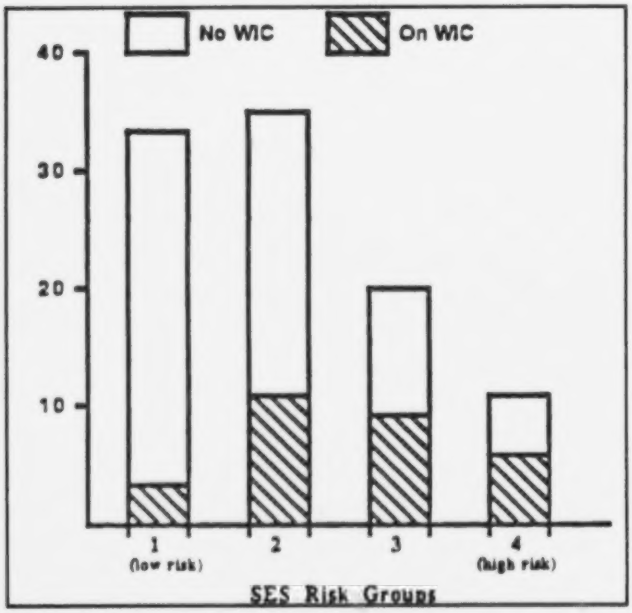


Figure 4: Distribution of all children born in Tennessee (total height of each column) , and all children enrolled in the WIC program (shaded portion of each column) among four SES groups. The highest risk group is represented by group 4 and the lowest risk group is represented by group 1. The relative proportion of children enrolled in WIC at each SES-risk group is also illustrated. Overall, the distribution of WIC children tends to skew toward the higher risk end, and a greater proportion of higher risk children were enrolled in WIC. However, the SES-risk background of WIC enrolled children crosses the entire SES-risk spectrum defined by this study.

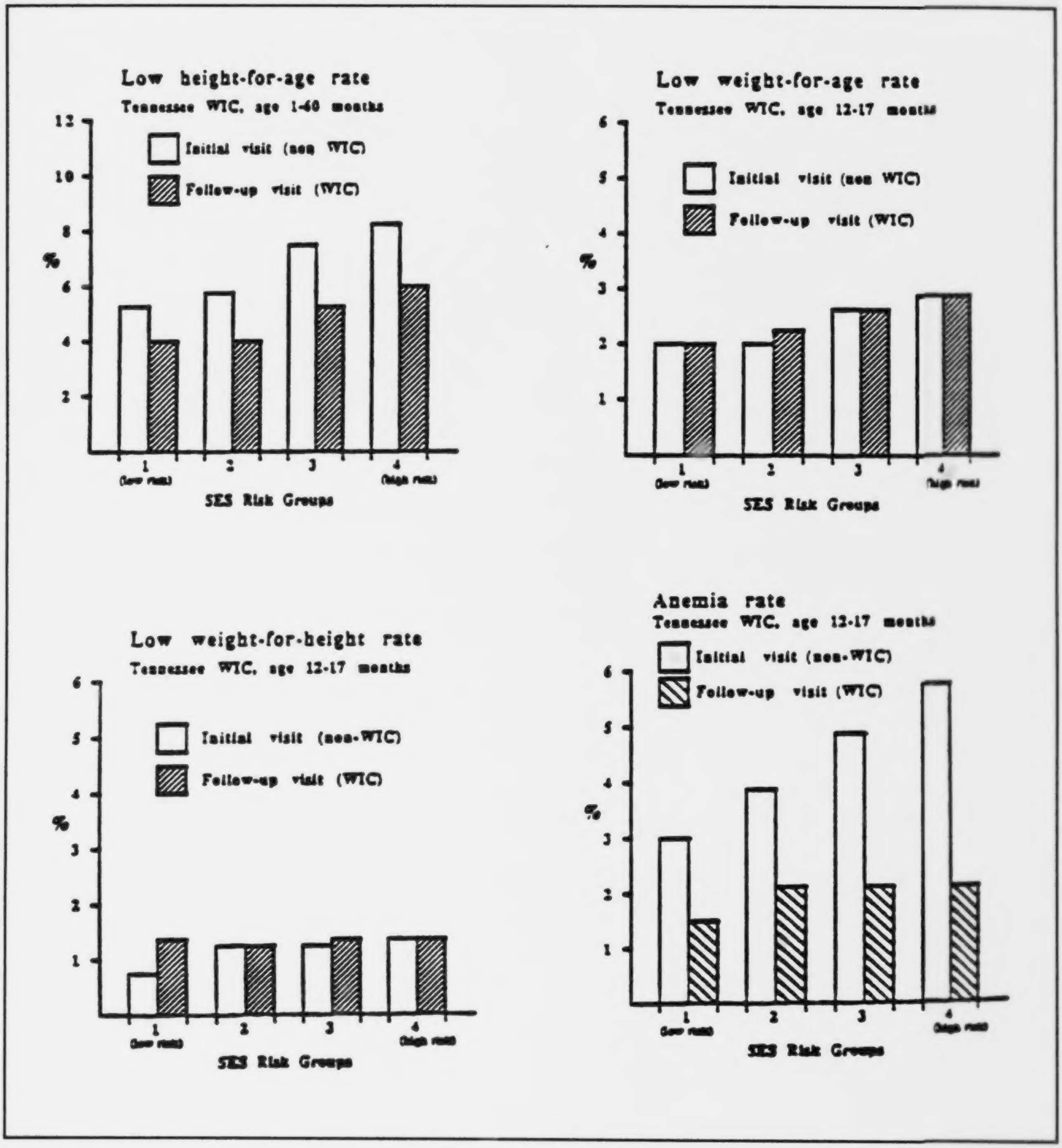

Low height-for-age rate
Tennessee WIC, age 1-60 months
Initial visit (non WIC)
Follow-up visit (WIC)
%
12
10
8
6
4
2
1 (low risk)
2
3
4 (high risk)
SES Risk Groups
Low weight-for-age rate
Tennessee WIC, age 12-17 months
Initial visit (non WIC)
Follow-up visit (WIC)
%
6
5
4
3
2
1
1 (low risk)
2
3
4 (high risk)
SES Risk Groups
Low weight-for-height rate
Tennessee WIC, age 12-17 months
Initial visit (non-WIC)
Follow-up visit (WIC)
%
6
5
4
3
2
1
1 (low risk)
2
3
4 (high risk)
SES Risk Groups
Anemia rate
Tennessee WIC, age 12-17 months
Initial visit (non-WIC)
Follow-up visit (WIC)
%
6
5
4
3
2
1
1 (low risk)
2
3
4 (high risk)
SES Risk Groups

Figure 5

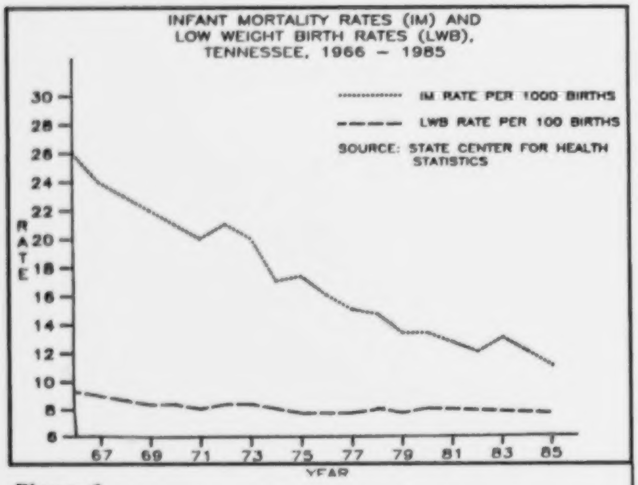


Figure 6

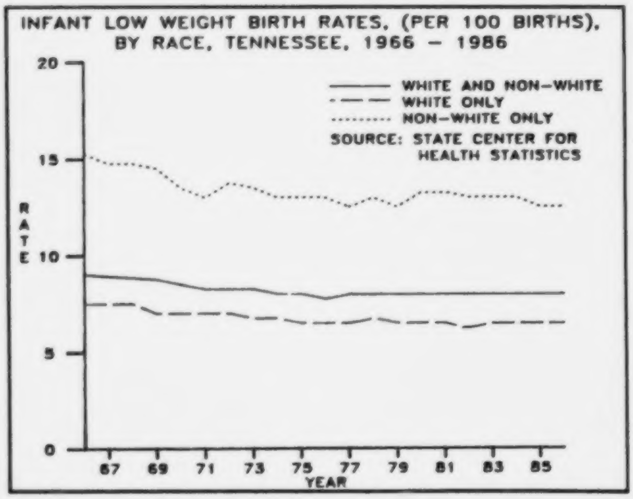


Figure 8

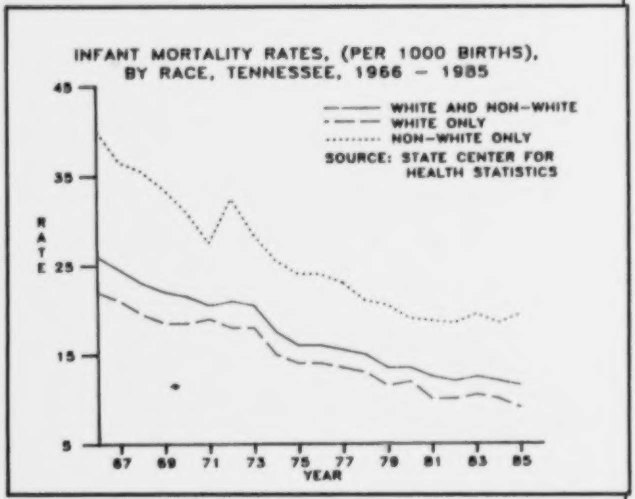


Figure 7

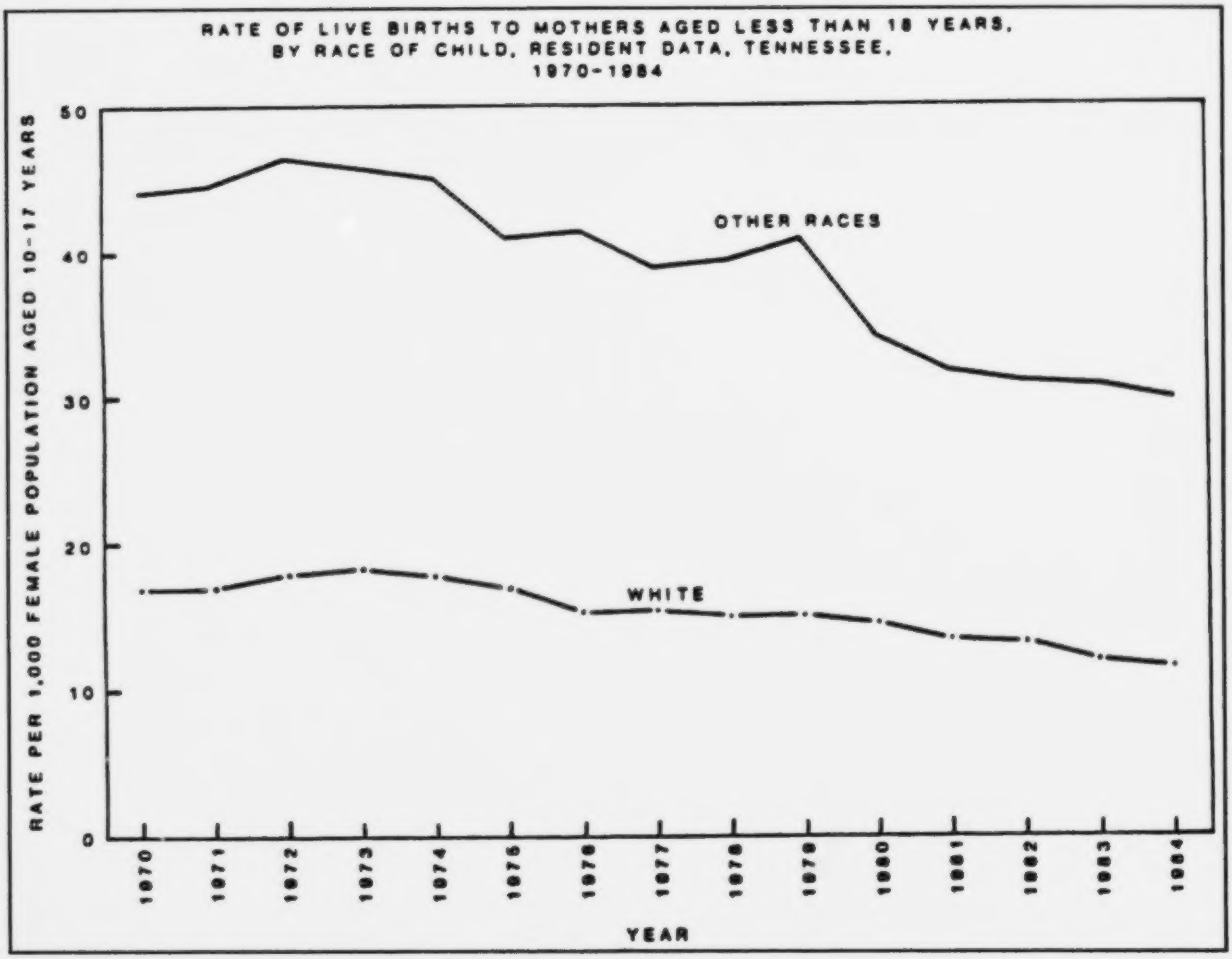

RATE OF LIVE BIRTHS TO MOTHERS AGED LESS THAN 18 YEARS,
BY RACE OF CHILD, RESIDENT DATA, TENNESSEE,
1970-1984
RATE PER 1,000 FEMALE POPULATION AGED 10-17 YEARS
50
40
30
20
10
0
OTHER RACES
WHITE
1970
1971
1972
1973
1974
1975
1976
1977
1978
1979
1980
1981
1982
1983
1984
YEAR

Figure 9

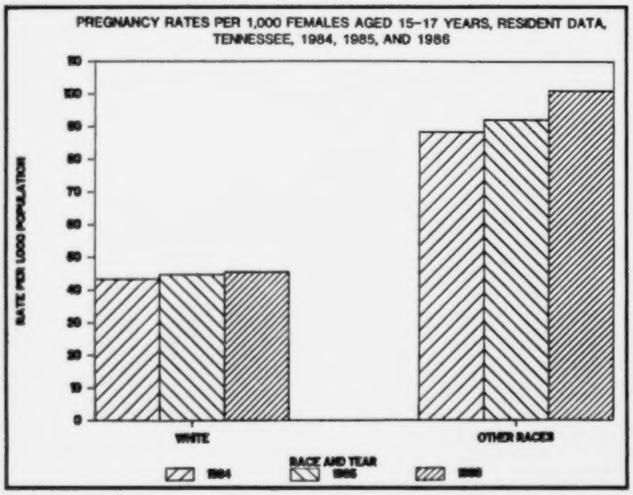


Figure 10

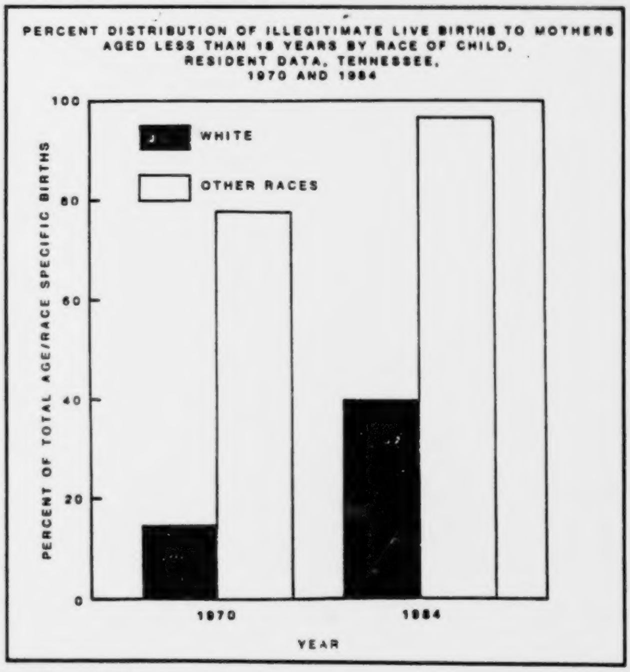


Figure 12

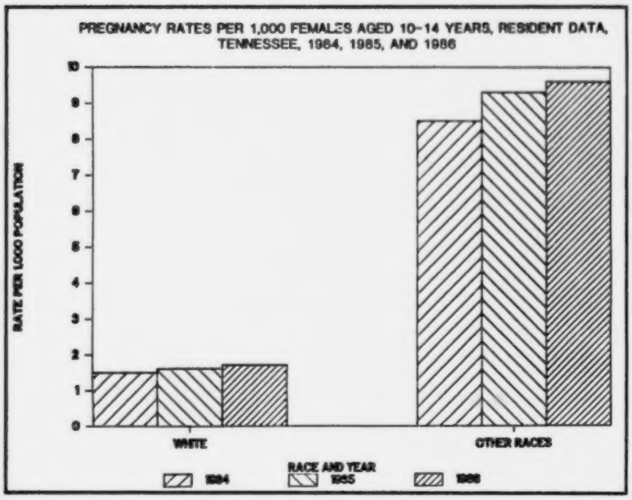
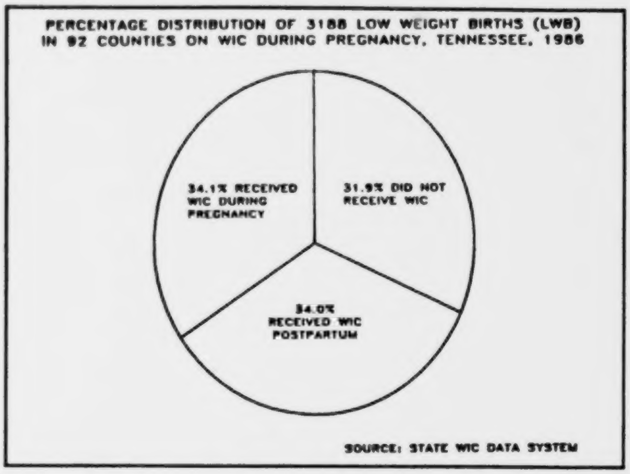


Figure 11



SOURCE: STATE WIC DATA SYSTEM

Figure 13

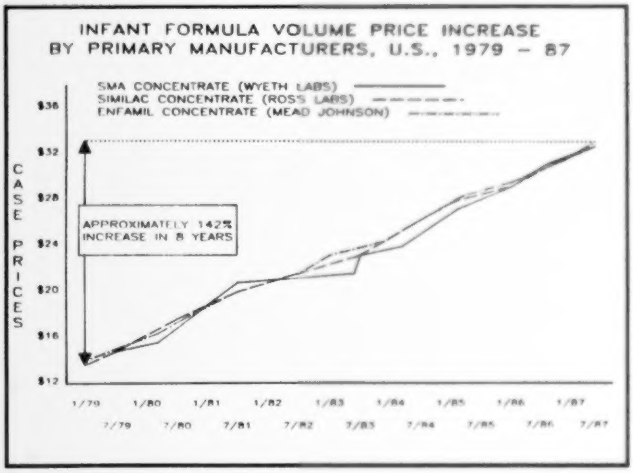
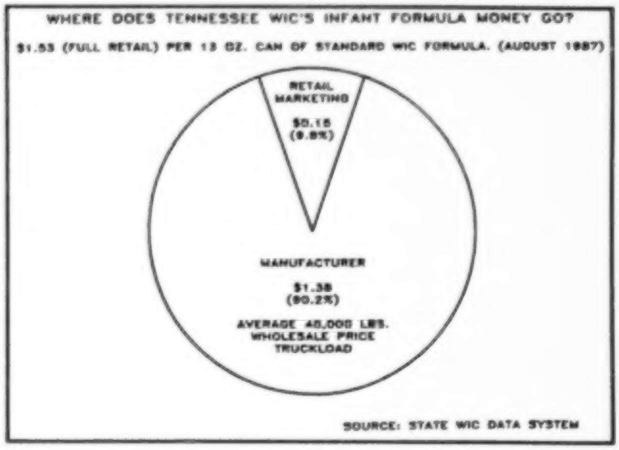


Figure 14



SOURCE: STATE WIC DATA SYSTEM

Figure 15

Beyond WIC: An Assessment of Nutrition Needs In Tennessee Maternal and Child Health

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Tennessee Department of Health and Environment
Nashville, Tennessee

Introduction

I first want to thank you for being asked to participate in this historic conference. The State of Tennessee and Meharry Medical College have long enjoyed a mutually supportive relationship, and I am particularly pleased to be able to speak to you today as both a state health official and a faculty member at Meharry.

The title that I have chosen for this presentation is "Beyond WIC: An Assessment of Nutrition Needs in Tennessee Maternal and Child Health Programs." The bulk of what I have to say is a partial report of a survey that was carried out this year by our Maternal and Child Health Section and the Nutrition Division of the Tennessee Department of Health and Environment. When I use the term, "Beyond WIC," I in no way wish to imply that the Supplemental Food Program for Women, Infants, and Children has not been effective. Quite the contrary. There is steadily accumulating evidence showing that the WIC program has had a tremendous impact on the health of mothers and infants in this country (1-4). But I use the term to try to remind myself and you as health care professionals that we cannot become complacent just because the WIC program has been so successful. There remains much to be done in assuring that all the women and children of our state have adequate nutrition. My report will help identify some of these pockets of need not currently served by the WIC program.

Relationship between Maternal and Child Health and Nutrition Services

The relationship between Maternal and Child Health and Nutrition Services established its roots before the turn of the 20th century. Nathan Straus, in New York City, established "milk stations" to help ensure clean milk to those in need. In 1912, a statute establishing the United States Children's Bureau was passed. Early Children's Bureau studies included such subjects as day care, working mothers, the nutrition of children, and the height and weight of children including those with heart disease, rickets, and malnutrition.

The Maternal and Infancy Act of 1920-29, also known as the Sheppard-Towner Act, emphasized economic aid to state and local groups providing health care, and helped pave the way for the provisions of Title V of the Social Security Act of 1935. This legislation was clearly one of the most outstanding legislative achievements of the 20th century affecting the health of mothers and children. It has provided federal funding to the states for prenatal care, well baby clinics, school health services, immunization services, health education, and public health nursing and nutrition services (5). Many other pieces of state and federal legislation have fostered the universal recognition that provision of nutrition and nutrition-related services has become a part of the core of what we do in public health, especially in our Maternal and Child Health Programs.

Tennessee Department of Health and Environment

Before I discuss our survey, let me tell you briefly where our MCH section and the Nutrition Division stand in the organizational chart of the Department of Health and Environment. The Department is comprised of 4 major Bureaus, including the Bureau of Health Services, the Bureau of Medicaid, the Bureau of Environment, and the Bureau of Health Manpower. The Bureau of Health Services, in turn, is comprised of 5 sections, namely Maternal and Child Health, Community Health Section, which includes the Nutrition Division, the Communicable Diseases Section, the Health Promotion Section; and the Chronic Diseases Section.

It is important to note that, organizationally, both the MCH Section and Nutrition Division are under the auspices of the Bureau of Health Services. Dr. Fleshood, the Director of Nutrition, and I, for example, both communicate directly with the Assistant Commissioner for Health Services. In addition to informal meetings between members of our central office sections, there is a central coordinating committee composed of staff from MCH, WIC, and Nutrition Services which meets bimonthly to discuss pertinent program issues. A policy of cross-training our program staff exists in order to enhance availability of services to our patients. For example, for the past 3 years our Crippled Childrens' program staff, along with Nutrition Services staff, have received training in special nutritional services to chronically ill and handicapped children. Finally, both MCH and Nutrition Services staff are members of the Child Health Standards Committee, which is charged with continually reviewing and updating the Child Health Standards Manual. This is a document which serves to measure the quality of care that is rendered to children in our public health clinics.

Our one hundred and eleven local health departments (LHD) are grouped into a total of 13 regions, and are supported by 13 Regional Health Offices. Each of the 13 Regional Office Directors reports to the same Assistant Commissioner for Health Services. While there is at least one public health nurse in every county health department, nutritionists are employed by the state, 12 of whom work at the regional level, and 71 of whom are assigned to the local level. Eighty percent of the funding for these positions comes from the federal WIC program. As you might guess, most of the nutritionists' time is spent in service delivery to WIC recipients.

Needs Assessment of Nutrition Services

Given the above information, there were several factors which prompted us in January of this year to plan a needs assessment of nutrition services in Tennessee. Although the state provided us with an "MCH-Nutrition Coordinator" position, the position was unfilled, and a thorough job description was not available. This had to be prepared. Secondly, the federal MCH office as early as 1985 was actively encouraging close cooperation between MCH and WIC at the state and local levels, and in August of 1986 published a report entitled "Improving MCH/WIC Coordination" (6) which we read and took seriously. Next, I had the opportunity to visit a neighboring state, and became aware of the fact that a nutritionist was a member of the program staff of each MCH program. Fourthly, I happen to be naturally suspicious of what is called "conventional wisdom." I believe that public health administrators can easily develop fixed and inflexible notions of optimal methods of health care delivery, and may fail to look closely at the most critical link in the entire delivery system, which I call the "system- patient interface." All our beliefs, our planning, our worthy intentions, and our spending is for naught unless the patient - the client, as some say - has his or her needs met in such a way as to function productively. Therefore, we chose to go to the "interface"-the local level-and depend on the experience of our local health department and school health nurses to try to answer two major questions:

1. To what extent are women and children in our Maternal and Child Health programs obtaining nutrition services unrelated to the WIC program?

2. To what degree is there integration of Nutrition Services with Maternal and Child Health programs?

Let me briefly describe the conditions of our survey, which is best referred to as an "hypothesis-generating" study. We wish eventually to translate the two questions I just mentioned into clear, precise hypotheses which can be tested experimentally.

The MCH Section is comprised of 8 programs: Genetics, School Health, Prenatal, Child Health, Crippled Childrens' Services, Perinatal, Speech and Hearing, and Family Planning. This survey was conducted only within the first five programs, and I will report today on the first two: genetics and school health.

Our target, as I mentioned, was "hands-on" providers. Survey instruments were prepared by MCH and Nutrition Services personnel, and were distributed to local health department and school health nurses in May 1987. A few caveats must be kept in mind:

1. Not all questions were answered by all respondents.
2. In a few instances, more than one answer was called for; these will be explained as the data are presented.
3. The data presented are largely nurses' estimates of service-related questions. Patients were not interviewed, and we have not as yet attempted to correlate the estimates with documented services received.
4. No statistical procedures have been applied to the responses.
5. My report today includes responses to selected questions. Time does not allow detailed responses to all questions asked.
6. Finally, this study does not attempt to address the quality of services.

The Tennessee Genetics Program was established by state statute in 1985. It provides operating expenses to six Genetic Centers throughout the state; to the Inborn Errors of Metabolism Laboratory at the Child Development Center in Memphis, the Comprehensive Sickle Cell Center at Meharry Medical College, and the Memphis Regional Sickle Cell Council. The program does not provide for direct reimbursement to providers. Services are delivered primarily at the above mentioned Centers, but health department patients who happen to be affected by genetic diseases can be expected to require health department services in between Genetic Center visits.

The intent of surveying LHD personnel was to discover what nutrition services were available routinely at the local level to patients with genetic diseases and their families. Survey questions were grouped accordingly to three topics: phenylketonuria (PKU), hemoglobinopathies (HGB), and others. Data from only the first two are presented. Seventy nurses returned the genetics survey instrument.

In response to the question, "Is there a staff member at your LHD who can answer questions directly related to PKU management?", 63%(43) answered "yes," and 37%(25) answered "no." Two did not respond. All who answered negatively, however, reported that they did have access to a nutritionist who could respond to PKU-related matters. Sixty nurses said they were not currently following PKU patients, and only 8 reported that they were currently doing so. Two did not respond.

Forty-seven of the 70 nurses responded to the question "In your community, who provides formula to PKU patients?" The 47 nurses provided a total of 84 answers, since multiple answers were possible. The rankings were as follows:

Local pharmacies	29
CCS	22
WIC	15
LHD	12
Other	6

We also asked nurses whether they had read reports from Genetics Centers regarding the status of PKU patients. Of the 70 nurses, 36 said they had done so, 18 said they had not, and 16 did not respond. Just over half of those who answered affirmatively said that they had incorporated nutritional recommendations from the Genetics Centers into their nutritional counseling.

We asked nurses how they would manage a woman in their LHD who is at risk of having a PKU-affected child. Sixty-four of the 70 nurses responded, and offered a total of 106 answers, since multiple answers were possible. Their answers are ranked as follows:

Referral to Regional Office Nutritionist	41	(39%)
LHD Nurse Counseling	29	(27%)
Referral to Genetic Centers	17	(16%)
Referral to CDC, Memphis	13	(12%)
Referral to physician	6	(6%)

Turning now to hemoglobinopathies, we wished to know whether our LHDs offered nutritional counseling to such patients and their families. Sixty-five nurses responded to the question. Thirty-nine (60%) said that they did provide counseling, and 26 (40%) said that they did not. The 39 nurses answering affirmatively provided a total of 43 answers when asked what kind of professional provided such counseling. "Nutritionists only" were listed 8 times; "nurses only" 6 times; "both of the above" were listed 24 times; and "others" were listed in 5 instances. Thus, responsibility for counseling was shared in most cases by nurses and nutritionists.

We also wanted to know whether LHDs were routinely offering nutritional counseling to prenatal patients with hemoglobinopathies. Sixty of the 70 nurses responded to the question. Of these, 27 (47%) said that they did so, while 31 (53%) said they did not do so. Ten nurses did not respond, and 2 gave non-categorical answers.

Finally, we asked whether the nurses perceived a need for materials detailing appropriate nutritional counseling for patients with hemoglobinopathies. Sixty-two nurses responded. Of these, 36(58%) responded affirmatively, while 25 (40%) said that they did not perceive such a need. One nurse said "maybe," and 8 nurses did not respond.

Opportunity was given for spontaneous comments or recommendations. These are summarized as follows:

1. Some nurses reported that they had never encountered a patient with PKU or HGB in their LHD.
2. Patients were considered "genetic" or "sickle cell" patients, but were seen and served according to their needs.
3. Regional Office nutritionists or regional medical centers often provided nutritional counseling for patients with genetic diseases.

We arrived at the following conclusions based on the genetics-related survey:

1. Because of the relatively low prevalence of phenylketonuria, most LHD nurses are unlikely to be currently providing nutritional counseling to families of such patients. Most (60%), however, reported having LHD staff capable of doing so and the rest reported access to a nutritionist who could do so. Thus, while nearly all PKU-affected families can be expected to have access to nutritional counseling services in Tennessee, such services should be provided as close to home as possible. Regular in-service regarding PKU would benefit LHD nurses and such families.
2. Careful inquiry needs to be made of PKU-affected families as to their source of formula and their ability to pay for it. Families qualifying for the WIC program for example, have incomes no greater than 185% of the federal poverty level. WIC families with PKU-affected children may find themselves suddenly having to bear the full cost of formula purchases when their child reaches the fifth birthday, and may need other resources.
3. The majority of nurses reported that a woman identified as being at risk for PKU offspring would be managed by referring outside the LHD. Study needs to be made as to what extent routine attempts are made to identify such women at risk, especially in the states' Family Planning Program. The knowledge base of all LHD providers may need to be broadened.
4. Nurses in West Tennessee and in urban health departments are more likely to see patients with hemoglobinopathies, which might explain the relatively low number (58%) of respondents in our survey reporting availability of nutritional

counseling to such patients. More information is needed on the content of such nutritional information.

5. Prenatal care is offered to women in every county of the state. The most basic care offered includes referral to WIC for nutritional counseling. Yet a majority of nurse respondents reported that routine counseling for prenatal patients with hemoglobinopathies was not offered in their LHD. More than half of those surveyed perceived a need for more educational material related to such conditions. Clearly, a need exists to provide basic nutrition-related information to all LHD prenatal patients who themselves suffer from HGB.

School Health Program

Let us turn now to the School Health Program, and the results of our survey of that program. The Tennessee School Health Program is commencing its second year of activity. The Basic School Health Program operates in 144 (6%) of Tennessee's 1,679 public schools, and potentially serves about 53,000 students, which represents 7% of the state's public school student enrollment of 843,754 (based on enrollment in December, 1986). The basic program currently operates only in grades K through 7, and is operative in at least one county in each of the 13 Health Department Regions. The aims of the program are to:

1. assist communities in identifying their own school health needs, and in developing community-specific responses to these needs;
2. work with schools to prevent as many student health problems as possible and to assure early intervention when problems are identified;
3. provide support to teachers in teaching the Health Education Curriculum currently mandated for Tennessee's public school system;
4. demonstrate the effectiveness of specific health promotion models in improving selected areas of health knowledge or health behaviors.

School nurse activities include, but are not limited to:

1. screening and referral for health related problems;
2. management of communicable diseases; and
3. consultation with school personnel in obtaining appropriate health care for children with special health needs.

Our nutrition questionnaire was sent in May 1987 to all 28 basic program School Health Nurses (SHN), and 100% responded. The nurses were able to provide specific information about free and reduced school lunch and breakfast programs as of February 1987. We wanted to know what proportion of students took advantage of each. We found that in the schools participating in the state's School Health Program, 19,500 students (40%) participated in the free school lunch program, and 3,400 (8%) took advantage of the reduced rate lunch. There were 11,800 students (22%) who participated in the free breakfast program, and 1,500 (3%) bought breakfast at a reduced rate.

We then asked the SHNs what barriers or problems had been noted for students in receiving the above benefits. Nine of the 28 nurses (35%) reported that buses arriving at school too late for children to have breakfast at school was a problem. Six (23%) said that the stigma attached to the programs was a problem. Two nurses reported that receiving breakfast interfered with classes. Nine (35%) listed various other problems, such as parents being willing to pay for reduced rate lunches, but not for reduced rate breakfasts; parents failing to return applications for the programs; and parents failing to bring children to the schools in time for breakfast.

We next asked whether the SHNs knew of students needing dietary modifications in their schools. Nineteen (68%) responded that they did know of such students, and 8 (29%) said they did not. One nurse did not respond. The nineteen nurses who responded affirmatively reported a total of 70 students who needed dietary modifications, which represents only 0.01% of the 53,000 total students, and is clearly an underestimate, based only on the estimated percentage of obese children (7). Nineteen of the SHNs (68%) reported that the school cafeteria was able to make menu substitutions that met specific needs. Three nurses reported that the cafeteria would not make substitutions, and that parents had to supply appropriate diets when needed. In one school, a cafeteria worker bought skim milk regularly for a diabetic child.

We wanted to know whether schools had access to trained individuals who were able to make necessary documented diet modifications to meet the needs of specific children. The majority (87%) said that such individuals were available, and 3 said that they were not. Food service managers were the personnel most frequently named, followed by Regional Health Office nutritionists.

Since one of the school health nurses' duties is to provide necessary health counseling, we asked how many children and/or parents (guardians) were counseled regarding diet relative to certain conditions during the past school year. Twenty-five nurses reported 119 such counseling encounters, and their frequency was ranked as follows:

Overweight	89
Anorexia	8
Bulimia	5
Food Allergy	4
Food Swapping	13

Three nurses reported that they had not engaged in any dietary counseling in the prior year.

Snacks and the quality of snacks may be important factors in the overall nutritional status of children. We attempted to assess this issue by asking whether there were food vending machines in the school to which the children had access. Seventeen (61%) nurses reported that such machines were available, and 11 (39%) said that they were not. In one case, there was a daily candy break under the direction of the principal. An ice cream vending machine was available to 7th and 8th graders in another school, and one junior high school had a concession stand which opened at the end of the school day.

The 17 SHNs reported the following types of food sold in the machines:

Candy	10
Chips	10
Coffee	1
Cookies	9
Crackers with/cheese or peanut butter	10
Juices	5
Milk	1
Nuts	7
Soft drinks	13
Pastries	6

Formal classroom teaching concerning health is also a responsibility of our SHNs. They reported teaching a total of 164 classes in the 1986-87 school year to do with nutrition subjects. Those topics most often taught were as follows:

Basic nutrition	21
Healthy snacking	12
Weight Control	8

Other topics reported were anorexia, bulimia, hypertension, food advertising, vitamins, foods harmful to teeth, and the importance of breakfast.

Types of nutrition services that SHN most often listed as needed in schools were:

1. More programs on weight control and choices of healthy foods (19).
2. Education about the effects of dietary sodium, sugar, fat, and caffeine (12).
3. Increased basic nutrition education at all levels (9).

A section was also provided for spontaneous comments and recommendations, and the responses were very interesting. SHNs thought that all cafeteria staff members should be encouraged to participate in ongoing hypertension control programs, and that cafeteria managers are and should be active in planning and implementing health promotion activities. Several nurses suggested that school cafeterias decrease the amount of fried food served, and increase the amounts of fiber. Finally, the recommendation was made that salt should be removed from cafeteria tables.

Conclusions

From the above survey, we reached the following conclusions:

1. In Tennessee schools participating in the MCH School Health Program, nearly half the students took advantage of free or reduced rate lunch programs, and about one fourth took advantage of free or reduced rate breakfast programs. These rates closely parallel those for U.S. school children as a group (8).

2. SHNs need to carefully explore barriers and problems associated with participation in such programs, so that no child who is in need goes unserved.

3. A very small minority of children has been identified to date as being in need of dietary modifications in participating schools. Enhanced efforts at identification and reporting of such children will no doubt result in a much greater proportion being identified. SHNs must help ensure that school cafeterias are then able to modify the diets of children who need such modifications.

4. Obesity in school children is a major source of concern to SHNs, and better strategies need to be developed to address this problem in Tennessee.

5. Efforts should be accelerated to substitute nutritious snacks to those schools for vending machines that presently dispense snacks with excessive sodium, sugar, and fat content.

6. Finally, school food service managers and cafeteria staff may be an untapped resource in some schools for inclusion in school health programs promoting healthy nutritional habits in school children.

Summary:

1. Nutrition services surveys which are carried out at the local level can be very important in helping program administrators determine what is actually occurring.

2. The "conventional wisdom" of program administrators may not adequately assess nutritional needs in children served in health department programs, and needs to be continually re-evaluated on the basis of epidemiologic data received from providers at the "system-patient interface."

13. Conclusions reached in this study help focus on areas of greatest perceived needs at the local level and can help form the basis for more scientific studies assessing documentation; patients' knowledge, attitudes, and practice; and quality of service rendered.

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NUTRITION ISSUES IN PUBLIC AND INTERNATIONAL HEALTH

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In looking through the program for this conference, I am worried that we may have fallen into a trap that Professor C.W. Churchman calls "the environmental fallacy", that is, the fallacy of ignoring the specific environment within which every problem is inextricably bound. By this he means that merely defining the problem and observing that there is a clear and urgent need to do something about the problem within a given system (in this case the "food" system) is not enough, particularly if the problem is assumed to be solvable within its own discipline. This denies access to potentially relevant or critical aspects of a phenomenon and fails to examine and compare the internal and external realities of the system which may be value and judgment laden. In other words, one must be constantly alert to whether the correct questions are being asked or whether the system is being properly defined, before plunging into potential solutions.

I would suggest that calling this talk "Nutrition Issues in Public and International Health" creates an environmental fallacy wherein the question is not put into the framework of a system, but has been reduced and "targeted" to suggest that "the problem" is nutrition issues and "the outcome variable" is health. (Parenthetically, I would point out that the title of these meetings also commits an "environmental fallacy" when it speaks of "Blacks and Other Minorities". It should address the impact of nutrition on the majority of the world's people, as well as the white minorities.) In any case, targeting nutrition as the problem or even a problem is a very narrow view of a complex set of issues that we all would agree are interrelated, and within a system where unidirectional arrows delineating cause and effect are without basis in "reality".

Having long ago switched my own perspective from the "reductionist and rationalist" approach of a nutritional scientist, and having attempted, in my analyses, to assume the role of a "participant", by placing myself in the cultural (if not actual) context of the poor and hungry, I find that the questions asked and solutions offered by my rationalist and disciplinary colleagues are often unsatisfactory and contradictory.

Let me elaborate slightly. What is the nutrition problem? In its simplest form, it is food accessibility – too little or too much. Obviously the problem of too little food can be solved by buying it or growing it. Simple? No! Are we justified in building a system to solve the problem of food availability that is only about food? I think not. The system of health is not broad enough to encompass the problem, but I will attempt to look at nutrition issues within the context of a health model which I would point out does not distinguish between health in the developing and developed worlds because the determinants of both are the same. They merely differ in degree.

This model, which is in no way unique, is clearly quite complex. So much that you may well ask whether this problem is really that complex or whether I am playing games to avoid taking responsibility for suggesting solutions. Actually, the situation is that complex, and I think that by pointing out these complexities we can begin to see why the public's health has deteriorated, instead of improving, over the last 50 or so years.How dare I say such a thing? Haven't our worldwide indices of health showed tremendous improvement? Hasn't life expectancy increased? Hasn't maternal and infant mortality dropped? Hasn't malnutrition decreased? Haven't sanitation, economic well-being and political democracy all increased? The answer to all these questions in many instances may be yes, but using these output criteria again raises the spectre of the "environmental fallacy". Are we asking the right questions? Are we examining "the numbers" correctly? Are these criteria, developed by the observers, but not the participants, laden with judgements?

I quote the following statistics (from the United States): (2)

* One of every two black children lives in poverty;

* One of every two black children grows up without a father;

* One of every two black teenagers is out of work;

* One of every four black births is to a teenager;

* One of every 21 young black men is murdered;

Add to this the fact that we do not know how many Americans are hungry or homeless and these figures do not offer much comfort in the most developed (and until recently, most economically self-sufficient country) in the world.

In looking at the determinants of health in my model, you will find that I have purposely not introduced health care into it since on its own, health care does not play a great role in health. The largest determinant of health is socio-economic status, regardless of what index is used (3,4). Obviously, then, the primary causes of inadequate food and ill health, both individual and public, involve inequities of various sorts and determining how they come about and what we can do about them is the problem we face.

If we go back to the problem of food accessibility and look at it on an absolute scale, throughout the world it has become worse. Let me show you some statistics from the 1986 World Bank Policy Study and discuss some of the reductionist thinking that went into their analysis.

What do the figures show us?

(1) that between 1970 and 1980 the absolute number of people in the world consuming energy deficient diets has increased and

(2) that about 770 million people live in countries where the energy content of food consumed was "low" in 1970 and by 1980 it had gone down further.

This, in spite of an optimistic statement on the cover of the report that "The world has ample food." What an indictment of our values and our "science.". (5)

Let us look at two separate (but still reductionist) partial explanations and solutions to these problems. Although the World Bank Report admits that the problem is complex, it attempts to single out the four most important conclusions to be drawn about food security. The first three are:

(1) "The lack of food security is a lack of purchasing power of people and nations..."

(2) "Food security does not necessarily result from achieving food self-sufficiency in a country..."

(3) "Food security in the long run is a matter of achieving economic growth and alleviating poverty...". (5)

The report then advocates cost-effective redistributive policies by governments to improve the food security of their people and the fourth conclusion addresses transitory food insecurity which I do not wish to deal with here.

We have heard these analyses and suggested solutions for almost 50 years: economic growth (for whom, not development in its broadest sense); governmental policies (established by whom, in mostly non-democratic settings) from above without ever asking the poor and undernourished themselves how they perceive the problems and solutions; the cry for trade, which has been and still is mainly exploitational for the Third World. These are not new "solutions". They have all been tried and have resulted in disaster after disaster. They are just being offered up again in new clothes, but we must ask why, if all the above "solutions" are supposed to work at the international level, are there still poverty and hunger in the U.S.? And what about all the other interrelationships in our model? In fact, let us try to examine the "consequences" of food insecurity. According to the World Bank Policy Study:

"The costs of inadequate diets to families and nations are considerable. Inadequate diets increase vulnerability to disease and parasites. They reduce strength for tasks requiring physical effort. They curtail the benefit from schooling and training programs. And they result in a general lack of vigor, altertness, and vitality. These outcomes reduce the productivity of people in the short and long terms, sacrifice output and income, and make it more difficult for families and nations to escape the cycle of poverty." (5)

Food insecurity causes all these problems so the answer is to somehow "target" the solution of getting food to the people who need it. There is practically no attention paid to the interactive features within the system which are also "causes" and the many complex feedback loops which are operational.

Let us look at another view, still reductionist, but broader. This was expressed by Dr. C. Gopalan in a monograph he prepared for WHO, entitled "Nutrition Problems and Programs in Southeast Asia" (6).

Gopalan suggests that health and nutritional problems in that part of the world are due largely to the tremendous backlog in development and the need to achieve what is usually referred to as rapid socio-economic advancement. He points out that the current world economic order is heavily weighted against them and "...even the development strategies which they would like to pursue are in some cases being inhibited and conditioned by the stipulations of international lending agencies from which they are constrained to seek help."

He goes on to provide a broader concept of development:

" 'Development', to be truly meaningful, must have as its central objective the improvement of the quality and status of the human resources of a country. Development involves two broad dimensions -- economic and social. Economic growth has to be accompained by equity and distributive justice if the benefits of such growth have to reach the poor sections of the population. Many countries of the Region have, for one reason or another, found themselves unable to address both these dimensions of development evenly and adequately, partly because of lack of resources and partly because of their own perception of their Plan priorities. Thus, for example, while some countries have achieved striking success with respect to overall economic development and augmentation of their food production, they have not been able to overcome poverty and food scarcity among large sections of their people with the result that small islands of plenty of coexist with vast pockets of dire poverty."

Here we see the emergence of a broader concept of development that entails both social justice and economic growth. But it still emphasizes economic growth and still involves an elitist top-down organization scheme for development.

It has been more than 40 years since President Truman first used the term "development" in its present sense (a defined method of social change). The concept has evolved from one of its original derivations of "unfolding" or social transformation, to one which allows for various input into the social change. Our suggestions for alternatives to the process of directed development will better address the health and nutrition problems of the underprivileged and malnourished of our society and other societies. But all we can do is draw up a blueprint. How to implement this is the more serious problem which I do not have time to consider.

I believe this can be put into more perspective if we examine briefly the argument of the philosopher, Paul Feyerabend who states:

"...that a free society will not be imposed but will emerge only where people solving particular problems in a spirit of collaboration introduce protective structures of the kind alluded to.

Citizen initiatives on a small scale, collaboration between nations on a larger scale are the developments I have in mind." (7)

We have heard before that authoritarianism goes with underdevelopment or that it could be a means of development and that development would then lead to political democracy (8). However, for the process of development to produce social transformation by collective decisions, it is necessary to create a truly free society. In such a society issues must be decided by open exchange -- the participants "get immersed into each other's ways of thinking, feeling, perceiving to such an extent that their ideas, perceptions, world views may be entirely changed. They become different people participating in a new and different tradition"(8). This contrasts with the usual guided exchange in which the participants are all rationalists and have power -- or, as Feyeraband states, "one has to play the game of the intellectuals." (7). A free society is characterized by Feyeraband as one "in which all traditions are given equal rights, and equal access to education and other positions of power" and must insist "on the separation of science and society" (7). In other words, we must remove the elitist authoritarian aspects of science and the scientist.

In conclusion, I would like to share with you some ideas that grew out of an interdisciplinary seminar of students, alumni and faculty at the University of California, Berkeley and San Francisco. The purpose of the seminar was to develop a better understanding of the determinants of health (and nutrition) and ways in which more effective action for achieving the visionary wish of health for all by the year 2000 might be fostered, if not achieved.

Health is not the same for all. Different people understand differently what health is for them, according to their culture, history, environment, social and economic organizations. That understanding also evolves within the dynamic context of their societies. General indicators, such as life expectancy, infant mortality and literacy are useful, but should not be assumed to have the same meaning for all, at all times. Less easily quantifiable indicators such as sustainability of development, rate of political participation, position of women in society, learning opportunities and cultural survival, are also informative and relevant.

Health is an individual right and a collective responsibility. Health arises within a complex web of human, social and ecological relationships. It does not exist separately from a healthy natural ecosystem, nor from peace, justice, collective intelligence and caring. Indeed, responsibility for ill health can often be traced back to political and economic violence and exploitation, inappropriate technologies, prejudicial policies and lack of social and human support. This is the justification for and meaning of intersectoral action.

Health is not guaranteed by economic lopment. Some statistics indicate a correlation between growth of GNP, a decrease in infant mortality, and an increase in average life expectancy. Many also agree that in situations of deprivation, increased availability of goods and service is health promoting. However, economic growth should not be equated with health improvements. Some countries with a lower GNP actually have better health status indicators than others with a higher GNP. And it cannot be denied that new health problems often arise in the process of economic growth, and that even in the most industrialized countries there persists a high level of ill health.

We believe that development should be a health-focused process of change. Accordingly to that stated above, such a process would be necessarily rooted locally, in the natural and cultural environment of the populations concerned, and unfold from the empowerment, rather than disempowerment of those whose lives it affects. We believe that the essential difference between a health-focused process of change and the usual "development" practices is that in the former the locus of control is at the grassroots. The implementation of this kind of development, which should not be restricted to developing countries, but rather pursued as a healthy change in developed countries too, requires coordinated efforts in different spaces.

The local community, both urban and rural, is the most appropriate and meaningful unit within which intersectoral action should take place. All individuals in a community live in a shared natural, social and cultural environment, and together experience various needs.

To be able to formulate and control health-promoting change, the community has to be aware of itself and its own potential power. Gatherings and community discussions of health problems and needs facilitate this process, and help people understand and support one another. Such meetings should be promoted, with particular attention given to the participation and concerns of the most vulnerable groups and minorities. Arising conflicts are a potential positive source of change in any community.

We believe that ministries of health should take a leadership role within national governments to allow and facilitate the process of local discussion of health-related problems and needs, to support the implementation of health projects decided upon by the local communities, and to be responsive to community decisions about management and allocation of scarce resources.

Ministries of health should work within their government, particularly in long term development planning, to assess the health consequences and health impacts of any large scale policy or intervention.

Ministries of health should persuade national governments to stop or modify programs, whenever these injure people, harm the natural environment or break fundamental social and cultural ties.

International promotion and support are imperative to carry out indigenously devised plans for change. In fact, WHO itself could be the best promoter of a new understanding of development as a health-focused process of change.

I fear that I have not offered you a clear-cut path to follow, but I believe that if we can continue to develop a dialogue in a spirit of exchange, respecting our tradition but realizing, as Feyeraband has so succinctly put it, they "are neither good nor bad, they simply are -- we may be on our way to achieve a more just and equitable world, one in which even the poorest, most deprived and disenfranchised would not participate for they would be no more." (9)

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Local and International Issues: Discussion

Stone, WL.

Dr. Fleshood, have you looked at the correlation between maternal hematocrit and low birth weight? There was a recent publication in the New England Journal of Medicine, suggesting that that was one of the best predictors of low birth weight regardless of socioeconomic status.

Fleshood, HL.

No, we don't have the hematocrits of the mothers.

Margen, S.

One of the two papers in the New England Journal of Medicine attempted to explain the differences in white - black infant mortality. The investigators looked at a whole series of variables and set up some very interesting, complex models. They made a very serious error in the papers. The error was that the hemoglobin and hematocrit of the mother remained constant from about the twenty fourth week of gestation to the end, and they cited an earlier paper to support this view. All other papers have demonstrated that women who are on any type of iron supplement have a significant rise of about 6 to 8 percent in their hemoglobin level during the last 3 or 4 weeks of gestation. If you correct for the latter, what the authors in the New England Journal of Medicine paper talked about would fall apart.

Rogel, AM.

My question is directed to Drs. Smith and Fleshood. You have both talked about the improvements in women and children as a result of WIC and the school lunch/breakfast program. Have there been corresponding changes in the way the students are learning at school? I ask this question in the context of the first day's presentation by Dr. Winick on how malnutrition affects the development of children.

Smith, JC.

No, I can't answer that. I have no data showing beneficial effects on student performance, specifically attributed to the school lunch/breakfast program. Most of the data are in other areas, for example, the adverse effects of lead toxicity.

Fleshood, HL.

In Tennessee, we don't have any data that I am aware of, which deal with the question that you are asking.

Smith, JC.

I think it is a very good question, and something that needs to be tested. What we have been doing in public health for many years is looking at process measures more than outcome measures. We need to begin to concentrate on the latter, and try to design studies which might specifically attribute a beneficial effect to one of our interventions.

Margen, S.

I am sure you are acquainted with the extensive evaluation of the school lunch program by the USDA, which was published in two volumes a few years ago. It was to be done in sections. One was the evaluation of the actual impact of the program on food intake. The second was on anthropometrics, and the third was on performance. They are still working on it. They are action programs and not research programs. We don't have a base line or control group. So, it is a formidable project.

Smith, JC.
I just might mention that one of the variables we are addressing in our school health program is chronic health related absenteeism.

Anonymous.
What is the incidence of PKU in Tennessee?

Smith, JC.
The same as anywhere else - about 1 in 15,000.

Anonymous.
Why are you particularly interested in your school nurses knowing about PKU?

Smith, JC.
No, not school nurses. These are local public health nurses. We are interested in it for several reasons. One, eventhough the incidence is low, we are presently following in our state program, between 40 and 50 people who have PKU. Some of these people are seen at genetic centers around the state, and some are not. Secondly, many of the female patients we are seeing are now reaching reproductive age. PKU patients were in years past taken off formula at age 4 years, and as you probably know, the recommendations are now to stay on the formula and have phenylalanine monitored throughout the adult years. Then thirdly, there is an ongoing cooperative US/Canadian maternal PKU study that has at least made us aware of the need for the states to begin to develop some kind of tracking mechanism for keeping up with the movement of people with PKU in and out of the states.

Herbert, J.R.
Dr. Fleshood, I was thinking that one way to reconcile the low birth weight data with data that you have on infant mortality might be to look into the distribution of low birth weights. In other words, you would expect the highest mortality rate in the very low birth weight group.

Fleshood, HL.
Well, we know of course that the state has about 5,000 low birth weights (8%) a year, and has had that for many years. The further away you get from the 2,500 gm mark, the smaller the numbers involved.

Anonymous.
Does that percentage change over time?

Fleshood, HL.
I have not really looked at that. Another point is that you can have services but if you are not getting the people to the services, then, you are not going to have the necessary impact. We need to look into why we are not getting them in. I was in Memphis recently and a woman in her seventh month of pregnancy came to the clinic at 7:45 am and left after 12:30 pm without getting her WIC voucher or nutritional counselling. We need to have our services designed for the patients (clients) and not for the doctors, otherwise the word is out there in the street that if a client (pregnant woman) plans to visit the clinic, she should be prepared to spend the rest of the day there.

Carter, JP.
I would just like to say that you get your women in and out in about half the time it takes to do the same in Charity Hospital, New Orleans. They get there at 7:30 am and are seen by the resident around noon. Most of the afternoon is spent trying to find out if they are eligible for WIC or supplemental food, and as a result, about 20 per cent of the mothers that deliver at Charity Hospital have had no prenatal care whatsoever.

The question I have is for Sheldon Margen. Short of restructuring the whole society, what do you propose that we do in the way of selling nutritional policy to improve nutritional status? Is there a role for nutrition planning - the so called, new applied discipline in public health?

Margen, S.
Your question is a very good one and I think it is possible as long as we have some sort of a democracy. There is still a possibility of changing our priorities and our social system. The two most important things that are necessary within the social change advocated are (i) alteration in terms of equity, particularly economic equity (ii) empowerment of the people who are now disempowered - the people who are disenfranchised and dehumanized. This is a job for every one of us. I don't think we need to have a revolution to achieve this but we certainly have to be aware of what needs to be done.

Smith, CL.
I have one question for Dr. Fleshood. The slide in your presentation - I am referring to the table that compares whites, blacks, and American Indians as far as prevalence of anemia is concerned - my question is why should native Americans be expected to have a lower prevalence of anemia? Is there any reason that we know of? There was a difference of as much as 50% in your slide and I was just curious about that.

Moragne, L.
I would be interested to hear the speakers comment on the notion of introducing an educational component as well as an employment component into food programs like WIC and Title V. We should encourage the women to continue their education or acquire some employment skills - something to really get the people on the road and off the programs.

Margen, S.
I think your question is right on target. This is why I say that for the problem of food accessibility to be corrected, we should really restructure the programs. Programs aimed at targeting only one single aspect of the individual's life will not solve the problems. The problems are embedded in a whole complex set of issues. I am not sure I would like to have our present congress do it.

Moragne, L.
How do you get these questions into the political agenda?

Margen, S.
That is the point. We have to work towards that so that we have programs which are not simply targeted for food or housing or welfare, etc. The programs have to be integrated so that the affected individuals are able to escape from the vicious cycle of dehumanization and poverty.

Smith, CL.
I think those are worthy goals for the future. My problem right now in our Title V programs is simply to be assured that the people who require the services get them. Crippled children for example; We had a 7 million dollar program and during the past year, 1.5 million was cut back for various reasons.

Closing Remarks

C. W. Johnson, M.D.,
Vice-President for Academic Affairs

You certainly have been inundated with knowledge over the past three days. There are only two or three kinds of things I would like to say. In the first instance I would like to read two sentences to you "continues its established partnership between college and community in promoting programs of health care, education and research; in light of its special concern for the health of minorities and the disadvantaged, Meharry through programs of research and through its health care service systems emphasizes the amelioration of health related problems of under served populations". Those two sentences are taken from the mission of the College. Missions are the sorts of things that people write and almost never look at. I just wanted to say that the past three days along with this nutrition workshop represent the articulation of the mission of this college at its best. I want to thank all of you for the part you have played in the articulation of that mission.

In the second instance, I would want to point out that some of you of course are visiting Meharry for the first time. You look at the buildings and you find an institution which has more than buildings as is the case in other institutions. You find the kind of a force and the kind of a spirit here which was presented by the focal on the staff and faculty and the students. Ronnie DeBoast talked about this thing in a book that he called "The God Within" and the phenomenon which he spoke of I called the spirit of place. It is the sort of thing that is based upon an institution's past, on its potential and on its future. Those of you who came to Meharry for the first time, have now been initiated into the spirit of this place. You are no longer strangers at Meharry. You are part of the extended Meharry family.

The third thing I want to say relates to Jim Carter's question and Doctor Margen's response as well as some of the discussions by the panelists. I was reminded of a book I read once titled "A Politics of Experience" by R. D. Lang. Incidentally, he is the only psychiatrist whom I read and I quote him to some extent. There is a little paragraph in this book where Lang says that what we think is less than what we know and what we know is less than what we love and what we love is less than what there is to love, and it is to that precise extent that all of us are less than what we are. You think of those words and think of the fantastic potential in this room. Thank you for being here and thank you for being.

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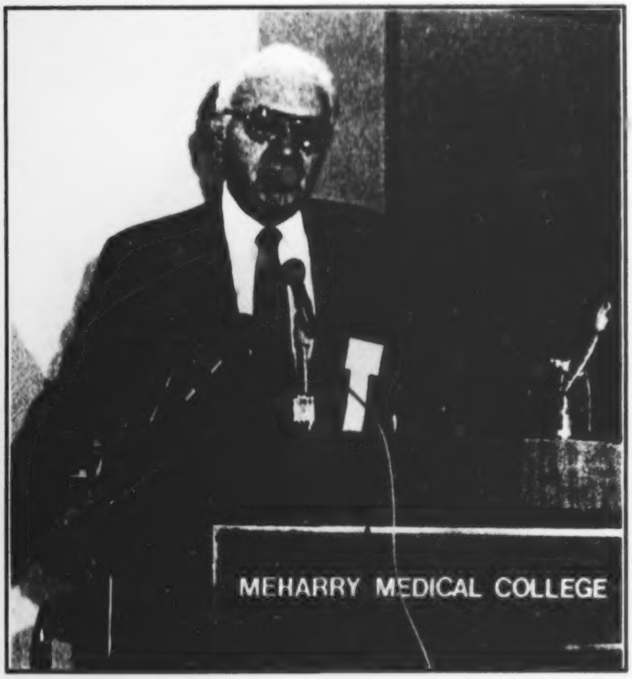
ACKNOWLEDGEMENT

Support for this Workshop was provided by The General Foods Fund, Inc. Additional financial support was received from the underlisted organizations:

Kraft Foundation
National Institutes of Health
The Philip Morris Companies
The Proctor & Gamble Company
Ross Laboratories
United States Department of Agriculture



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